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BRANCHIOGENIC CANCERS AND OTHER CARCINOMATA OF THE NECK OF CRYPTIC ORIGIN.

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CARCINOMATOUS deposits in the glands of the neck are of frequent occurrence. Those due to squamous epitheliomata in the oral cavity and adjacent parts, or to carcinoma of the thyreoid, or to carcinoma of the stomach *et cetera* do not concern the present communication. Occasionally carcinomatous growths are met with in the deep tissues of the neck and careful clinical examinations throughout the course of the case fail to reveal a primary growth in the mouth, larynx, œsophagus *et cetera*. A number of these growths are squamous epitheliomata, but in some cases the growth, though carcinomatous, is not obviously derived from squamous epithelium.

The object of this communication is to discuss the nature and consider the possible origin of these carcinomatous growths. During the last four years (1930, 1931, 1932, and 1933) 38,725 patients have been admitted to the wards at the Adelaide Hospital. During the same period 2,287 patients have been referred to the radium clinic. A number of the latter patients were not in-patients. But all the patients with carcinoma of the neck of cryptic origin amongst these radium clinic patients were admitted to the wards, and probably represent most, if not all, the examples of those with this type of tumour in the 38,725 in-patients. The cases comprise fifteen in which the source of the neoplasm

remained cryptic, and one case in which the origin was revealed only at the *post mortem* examination. The sixteen cases can be grouped as follows:

1. Definite squamous epitheliomata, usually with well marked cell nests: seven. The growths were not near the mid-line.
2. Squamous epitheliomata with cell nests, the growths under the jaw or near the mid-line: two.
3. Epitheliomatous, but the cells presenting a transitional appearance or suggesting a basal cell type: three.
4. Epithelioma suggestive of an origin from a nasal sinus: one.
5. Squamous epithelioma secondary to a growth surrounding and destroying the pituitary body: one.
6. Diffuse carcinomatous growth, possibly of epitheliomatous origin: one.
7. An indefinite neoplasm, possibly an endothelioma: one.

Details of these cases are given at the end of this paper, arranged in the order just mentioned.

Possible Origins of the Tumours.

A common explanation of the occurrence of some at least of these growths is that they are derived from the epithelium of a branchial cleft. Some ridicule such an origin, talking of "the so-called branchial cleft carcinoma of the ignorant".[2] It is necessary to consider in detail the possible origins of all deposits of squamous epitheliomata and other carcinomata in the neck before dismissing as untenable remnants of the branchial epithelium as the source of some of them. The following occur to us as probable or possible origins for such malignant cells:

1. In the majority of cases the malignant growths in the neck are clearly secondary to recognizable primary growths in neighbouring structures, or sometimes from those at a distance. Carotid body tumours have characteristics of their own and are not considered.
2. In or near the mid-line or close under the jaw growths may arise from remnants of the thyreoglossal duct or from outlying portions of a duct of a salivary gland, but by the time advice is sought, the size of the growth precludes a recognition of such an origin. Thus Case VIII (Radium Register 585) may have arisen in the lingual duct.
3. Remnants of the branchial epithelium left behind during development. We think it highly probable that our Cases I to VII are derived from this source, and perhaps also Cases X, XI and XII.

Ewing[4] recognizes branchiogenic carcinoma. Bland Sutton,[1] on the other hand, denies its existence, inferring that the supposed cases are really secondary deposits from small and overlooked growths elsewhere.

We think that there is no doubt at all that occasionally cases of carcinoma in the neck may originate from remnants of the branchial epithelium; in fact, the total absence of such carcinomata would be remarkable. The occurrence of branchial cysts is an indication that the branchial clefts may be imperfectly closed. Branchial cysts are not very common. During the last ten years the records of the Adelaide Hospital show that five cases (one perhaps doubtful) were operated on out of a total number of 86,364 in-patients. Possibly further cases occurred but have been indexed under other categories.

Boyd[3] states that the cysts are usually derived from the third branchial cleft and are situated at the level of the hyoid bone. If the cyst arises from the second cleft, it is situated just below the mastoid process and may project into the mouth. Boyd adds that, if congenital, they are usually not noticed till puberty. They are lined by either stratified epithelium or, if derived from the deeper portion of the cleft, by ciliated columnar cells with mucoid contents.

MacCallum[6] points out that the epithelium may ramify so as to extend far up behind the ear and downwards in the neck. He says that the cyst is usually situated somewhere between the mastoid process and the sterno-clavicular joint, along the anterior border of the sterno-mastoid muscle.

The four undoubted cases of branchial cysts seen in the Adelaide Hospital occurred in patients aged from eighteen to twenty-five years. Three were in young women. Dr. Bull examined the cysts removed and found that they were lined with squamous epithelium with lymphoid tissue in close proximity to it. The fifth and doubtful case was in a man aged seventy; in this instance the cells were thought perhaps to be endothelial. It seems, then, that branchial cysts are not common and that they usually manifest themselves, as Boyd has pointed out, at or near puberty. Why they do not appear before this age is not clear. In a closed cavity the shedding of the oldest epithelial cells lining the cavity should cause a gradual enlargement of the space. Possibly during puberty there is greater activity in the epithelium—we see this in the development of the beard in males—and such may explain the first appearance of recognizable cysts at or some time after this period. It is quite likely that, apart from the formation of actual cysts, islands of epithelial cells may be left behind as the branchial cleft closes, and that late in life these dormant cells may be stimulated into activity and be responsible for branchiogenic cancers.

In connexion with the teeth, we meet with dental cysts. These are lined by squamous epithelium and are surrounded by plasma cells. They are believed to originate from irritation, usually caused by infection, of groups of epithelial cells left behind by the enamel organ during development. A recent alternative theory is that the cysts are really the ends of sinuses lined by epithelium which has descended from the gum surface. The first view given above is that islets of epithelial cells may remain dormant for years, but that certain kinds of irritation may induce the cells to start multiplying. Observers have repeatedly reported the occurrence in the periodontal membrane, near the roots of teeth, of groups of cells, called after their discoverer the "rests of Malassez", which they consider with reasonable

probability to be quiescent remnants of the enamel organ or dormant buds from the epithelial tooth band. It is conceivable that squamous epithelioma may occasionally start in such epithelial remnants; these cases must, however, be rare, though possibly examples are overlooked by the early involvement of adjacent parts. Applied to the present discussion, in which the occurrence of branchial cysts is generally admitted, we may infer the probability that, apart from such a cystic development in remnants of the branchial epithelium, nests of dormant cells may also be expected to occur, and that in these, living under abnormal and doubtless disadvantageous conditions, malignant change is occasionally to be expected.

A frank squamous epithelioma may rarely develop in the squamous epithelium lining a relatively innocent cyst. The pathological museum at the Adelaide Hospital contains two examples of dermoid cysts of the ovary, in which the cavity is partly filled by a squamous epithelioma that also infiltrates the wall. In one the cystic space is nearly obliterated by the mass of growth. We know of no definite examples of such an event occurring in a branchial cyst. Such cysts, when detected, are usually removed, so that the time factor, which is of importance, cannot operate. It is possible that a small and unrecognized branchial cyst of long duration may at times be responsible for a squamous epithelioma, the mass of growth soon obliterating the cavity and a solid growth of cryptic origin resulting. Some presumed branchiogenic cancers are cystic, probably from secondary degeneration, though none of ours obviously presented this feature.

As regards the histology of the growths attributed by us to the branchial epithelium, we consider that the type most likely to be met with, in conformity with the nature of the lining wall of most undoubted branchial cysts, is a squamous epithelioma with or without cell nests. From this we think that departures may occur, so that transitional types may be met with from time to time approaching the basal-cell carcinoma, or even, as a rare occurrence, a diffuse infiltration with carcinoma cells, which may only here and there suggest an origin from a stratified epithelium. It is also possible that growths originating from the deeper portion of the cleft may show columnar mucin-secreting cells, though we have not recognized such.

Dr. L. B. Bull, by whom most of the sections from these cases were first diagnosed, laid stress on the fibrosis accompanying those growths that he believed had an origin in the branchial epithelium, and on the absence of any lymphoid tissue which might have suggested a deposit in a lymph gland. As regards the fibrosis, though this may not be a common feature in definite secondary deposits in the neck, it does occur at times in slowly growing masses. As regards the absence of lymphoid tissue, this would be expected to disappear eventually in a deposit in a lymph gland when growth had extended well beyond the original limits of the gland itself; and contrariwise, lymphoid tissue seems a frequent, if not invariable, accompaniment of branchial cysts and so might be expected to be associated with the remnants of branchial epithelium from which the branchiogenic carcinoma started, and further, the branchiogenic cancer may itself give rise to deposits in adjacent glands. Thus, whilst laying considerable stress on Dr. Bull's opinion, founded on a very extensive experience, we would bear in mind the points we have raised, which doubtless he did.

Incidentally, we might here call attention to the close association of lymphoid tissue with the epithelium in structures originating in the branchial clefts. We see this association invariably in the tonsils and in the thymus, frequently, if not always, in the cells of branchial cysts, and quite often in the thyreoid gland.

4. The possibility must be considered of the healing of small primary growths, some of whose cells may have escaped previously into the draining glands. Though it is perhaps possible for a small epitheliomatous ulcer to undergo healing from destruction of the malignant cells by fibrosis, the likelihood of this occurring must be very small. In accessible sites healing of malignant growths probably does occur from time to time. Some of us have seen the cure of a presumed rodent ulcer following the application of the juice of the spurge, *Euphorbia peplus*, and other unrecognized factors, apart from fibrosis, may also be operative. The likelihood, however, of such a spontaneous cure occurring in a small epithelioma in the oral cavity, some of the cells of which have first been able to escape and pass along the lymphatics, is very small. We think that such an origin must be dismissed as too theoretical for practical purposes.

Nevertheless we have had recently two examples of the widespread dispersal of carcinomatous cells in lymphatic glands in which a careful search *post mortem* failed to indicate a primary focus. These cases are being reported in an early number of *The Journal of the Cancer Research Committee of the University of Sydney*. All likely sources of origin were examined. We are left with two alternatives—either the primary focus had healed, or a small, but old, primary growth escaped detection.

The last-named view, that a primary growth is present but remains so small and insignificant that it escapes detection, or is situated in such a position as to be revealed only at the *post mortem* examination, is the one that appeals to Bland Sutton and others. An occasional case may be so explained.

In our series Case XV is an example of a primary growth situated out of ordinary reach and not revealing itself by obvious clinical signs. Squamous epitheliomatous glands appeared in the neck. The patient attended the radium clinic in Sydney and later that in Adelaide, but it was only after death that a mass of growth was found in the neighbourhood of the pituitary gland, and the growth was finally diagnosed as having had its origin in the epithelium of Rathke's pouch.

We think it likely that, from time to time, small growths in the recesses of the oral cavity and its adjacent structures do escape detection, in spite of close clinical observation over considerable

periods, but that such a source explains only a moiety of the cases discussed in this paper.

5. Another possibility which has to be considered is the introduction of cells from the Malpighian layer of the skin into the lymphatics as a result of trauma; for instance, puncture wounds by spicules of bone during eating. From time to time we see in the subcutaneous tissues examples of inclusion or implantation cysts resulting either from nipping off of a little epithelial fragment during surgical operations or from pushing into the tissues, as, for instance, by the blunt end of a needle, of a little group of cells from the deeper layer of the surface epithelium. It is possible that such puncture wounds may sometimes carry epithelial cells into the lymph spaces, whence they may be carried to the draining glands and there grow. We have seen a small epithelial-lined cyst in the axilla which may have had an origin in this way. In such an abnormal home the cells will be living under disadvantageous conditions, and this would predispose to a malignant change in them. The origin of a squamous epithelioma of the neck in this way, if it ever occurs, must be so rare that it loses all practical significance.

Two Examples of Subcutaneous Nodules Formed by Calcified Squamous Epithelial Cells Surrounded by True Bone.

We should, however, like to take this opportunity of recording, under a special heading, the occurrence of a rare condition in which sheets of calcified epithelial cells surrounded by true bone form hard subcutaneous nodules in the tissues of the neck and probably elsewhere. This is the *"nævus ossificans"* of Flarer,[5] and includes those cases of so-called "calcified epithelioma" in which true bone does not occur.

About 1908, the late Dr. Seed, of Perth, Western Australia, submitted to one of us (J.B.C.) some material from the subcutaneous tissue of the neck, which proved on examination to consist of sheets of calcifying epithelial cells surrounded and imprisoned by laminæ of true bone. At the time these epithelial masses were thought to be portions of a squamous epithelioma which was undergoing spontaneous cure from strangling of the cells by the surrounding mesoblastic reaction.

Quite recently, in Adelaide, a piece of tissue, also from a subcutaneous position in the neck, was submitted to Dr. Bull and presented an appearance exactly like that of the Perth specimen. Dr. Bull informs me that he remembers having had a third case, but we have been unable to find the section.

The recent example was in a man, aged thirty-five years, who had noticed a shiny tumour behind the angle of the mandible on the left side for about ten years. On examination there was found at the site indicated a hard, rounded tumour, 1·5 centimetres in diameter, with a small central ulcer exposing a hard white mass, which appeared to have burst through the skin. There was no deep fixation (see Figure I).

This is evidently an entity and not what might be called a freak occurrence. The sheets of epithelial cells are extensive and, owing to the calcification which has taken place in them, can in places only just be recognized (Figure II). The epithelial masses seem always surrounded by true bone which contains some osteoclasts. The condition is probably to be explained by the displacement into the subcutaneous tissues of small portions of the surface epithelium. The graft has developed in its new situation and, instead of forming a cyst, has produced sheets of cells which have become surrounded

FIGURE I. Showing the site and size of the nodule composed of calcified epithelial cells surrounded by true bone. Male, aged thirty-five years.

by dense fibrous tissue. Then, possibly due to the poor nutrition of the epithelial cells, calcium salts have been deposited in them, and this calcification has led to a similar change in the surrounding stroma, with the formation of true bone. The condition would seem to be analogous to the calcification that takes place in the cells of the enamel organ; this calcification is promptly followed by a somewhat similar change in the underlying mesoblast, which thus produces the dentine, which is a modified bone. These subcutaneous calcified plaques illustrate the tendency for epithelial cells to undergo calcification and show the way in which teeth, with their enamel and dentine, in all probability originated.

Clinical Discussion.

It is, of course, natural that all the cases of cryptogenic carcinoma recorded at the end of this paper should have been carefully investigated. None has escaped the attention of a consultant oto-rhino-laryngologist, and in most cases radiographic investigation of the lungs and nasal accessory sinuses has been carried out in addition. In no case has the œsophagus been thoroughly examined, but

symptoms have not suggested the necessity of such examination, and it is a fair statement that, if present in the regions suggested, a primary lesion must have been of insignificant dimensions.

If an occult primary tumour in the mouth or naso-pharynx were present in any considerable proportion of these patients, it is only reasonable to expect that occasionally one would make its presence felt during after-progress. This has not occurred in any of our fifteen patients, the history of whose lesions extends over periods varying between seven and twenty-eight months, with an average of 13·6 months. It is rather unlikely that deep X ray treatment would so consistently have healed or retarded the progress of an occult primary lesion, if present; and we have regarded the absence of late symptoms referable to such an occult growth as indicating the primary nature of the cervical tumours discussed. All of our patients have been males, and all but one clean-shaven; that one exception is the man whose nasal accessory sinuses were suspected. If repeated trauma during shaving were a feasible cause, it would be expected that a large proportion of the growths would appear in the submaxillary glands, and this has not been the case.

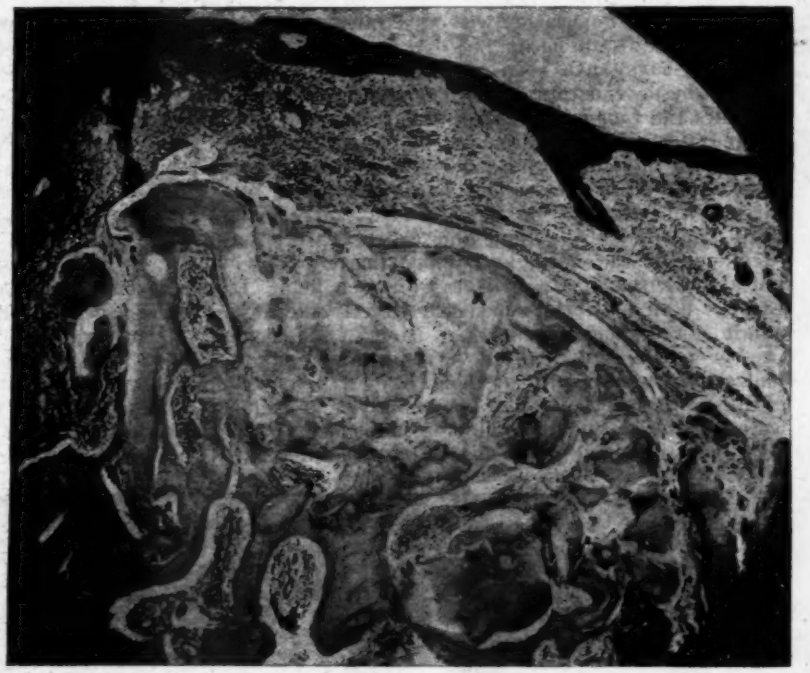

FIGURE II. Low power section, showing surface epithelium and below this the nodule. The bony trabeculæ surrounding the epithelial cells can be clearly seen. There are many osteoclasts round the edge.

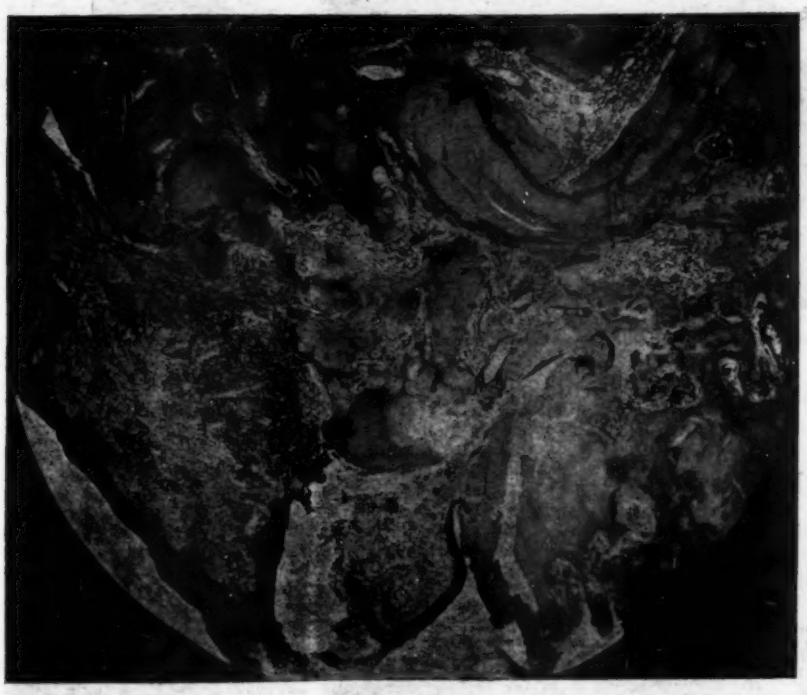

FIGURE III. Similar to Figure II, but showing the nodule ulcerating through the skin.

Consideration of the cervical lesion shows that none of these patients was frankly operable, and a wide block dissection would have offered reasonable prospects of cure to very few of them. In the main, therefore, treatment has consisted of deep X ray therapy, combined with unilateral block dissection when the latter has appeared feasible.

The results of treatment have been consistently bad. All but three patients are dead, and all of those living offer possible or certain evidence of recurrence. Palliation, however, has been obtained in many cases, and even the mental satisfaction to the patient derived from observing partial resolution of his tumour has made at least some of the treatments worth while. One patient died of intercurrent disease seventeen

months after his operation without clinical evidence of recurrence in his neck.

The X rays have been produced from a tube excited by a constant potential machine at 200 kilovolts and four milliampères of current, and were filtered through 1·0 millimetre of copper and 0·5 millimetre of aluminium. The size of field has been either 80 or 150 square centimetres, and the target skin distance 40 centimetres. Six fields were irradiated, two oblique and a direct lateral on each side of the neck, and approximately 900 *r* units, measured in air, delivered on each field. It has not been feasible to measure directly the dose administered, because the apposition of the compressor to the skin precludes the insertion of an ionization chamber into the field.

In several of the earlier cases only four fields were irradiated, and the whole of one aspect of the neck was included in each field. The effect of irradiation has been to produce aphonia and dysphagia, with bright red erythema of the skin in one to two weeks, followed by mild epidermolysis and deep bronzing. A leathery skin has remained after about eight weeks.

Discussion of the site of origin of the tumours is rendered difficult by the fact that most of the patients have died out of hospital and autopsies have been conspicuously absent. Even at autopsy, however, it might be quite impossible to indicate any particular cleft when a tumour four to eight centimetres in diameter occupies a position in which three or four of the branchial clefts fuse, and it is still more difficult clinically to arraign any one of the clefts. The most accurate statement that can be made is that many of the tumours may arise from the second, third or fourth clefts, while those tumours more anterior in position probably cannot be connected with the fourth cleft.

Cases Classified in Groups.

Group I: Squamous Epitheliomata, Often with Well Marked Cell Nests, Probably Arising from Branchial Epithelium.

Seven cases (Cases I to VII), all occurring in males, aged 51, 56, 56, 56, 66, 70, 72 years of age, fall in Group I, that of squamous epitheliomata, often with well marked cell nests. Their origin is best explained as from the branchial epithelium. In six cases the tumour was mesial and anterior to the upper portion of the sterno-mastoid muscle. In one case the tumour was situated more anteriorly, practically occupying the submaxillary triangle.

FIGURE IV. Higher magnification, showing the calcified epithelial cells still recognizable in the centre, surrounded by true bone.

CASE I (registered number 610).—M.L., a male, aged fifty-six years, noticed a painless lump in the left side of the neck in April, 1930. Subsequently his voice became hoarse. Examination revealed a fixed mass of matted glands beneath the upper third of the left sterno-mastoid muscle without a recognizable primary tumour in the mouth, pharynx or larynx. The mass was excised in September, 1930, and Dr. Bull reported: "Sections show a squamous cell epithelioma." A radium mould, three centimetres in thickness, was applied to the left side of the neck in October, 1930, but there was no improvement and the patient died in March, 1931.

CASE II (registered number 1060).—R.J.P., a male, aged seventy years, was admitted in September, 1931, with a

large, tethered, fungating ulcer in the left submaxillary region, measuring five centimetres in diameter. He had noticed a lump at that site one year previously and six months later it had been excised in the Broken Hill Hospital. Sections showed deposits of a squamous cell epithelioma with cell nests. Examination in the Adelaide Hospital failed to reveal a primary tumour in the mouth, pharynx, or lungs, and a course of deep X rays was given at the end of September. He died two months later without showing improvement. An autopsy was not allowed.

CASE III (registered number 1390).—R.A., a male, aged seventy-two years, noticed a lump in the left side of his neck in April, 1932, together with tenderness at the roof of his mouth. On examination one month later there was a stony-hard mass infiltrating the upper half of the left sterno-mastoid muscle, and fixed firmly. Within the mouth was seen fairly extensive leucoplakia. The Wassermann test yielded no reaction. Dr. Bull reported on a biopsy specimen from the hard palate: "Sections show epithelial hyperplasia and hyperkeratosis, forming leucoplakia. There is no evidence of any malignant new growth."

A subsequent section of the mass in the neck was reported on as showing a squamous cell epithelioma. A primary focus could not be discovered elsewhere in the mouth, pharynx, or lungs. Deep X ray therapy was given in August, 1932, followed by improvement in the leucoplakia, but without much alteration in the tumour of the neck, and a second course was given in January, 1933. The tumour remained stony-hard until his death, which occurred in July, 1933, from hæmorrhage through a sinus which had persisted at the site of the biopsy wound.

CASE IV (registered number 1437).—D.McG., a male, aged fifty-six years, noticed a painless lump on the right side of the neck in April, 1932; it increased in size for several weeks and then remained stationary. On examination there was found a hard tumour, four centimetres in diameter, in the posterior triangle on the right side of the neck above its centre. The only abnormality to be found within the mouth and pharynx was a slight fullness in the right pyriform fossa without either irregularity of the surface or limitation of movements of the larynx. A radiograph of the lungs was normal. On June 4, 1932, a Crile's dissection of the right side of the neck was carried out, followed by deep X ray therapy in June and October, 1932. Dr. Bull reported: "Sections show a squamous cell epithelioma. There is a small lymph gland associated with the mass, but the tumour does not appear to be a deposit in a lymph gland."

This man remained well until November, 1933, when he died following an operation for volvulus of the small intestine. An autopsy was not allowed, but clinically there was no evidence of recurrence of the tumour, nor had any change in timbre of the voice been noted.

CASE V (registered number 1863).—S.A., a male, aged fifty-six years, noticed a small lump on the left side of the neck in August, 1932. This grew slowly and he developed pain in the left side of his head before his operation, which took place on March 25, 1933. At that time there was palpable a superficial mass attached to the skin behind and below the angle of the mandible on the left side, with a second mass deep to the sterno-mastoid muscle. A primary focus could not be found in the mouth, nose, pharynx, or lungs. Incision revealed a "collar-stud" abscess, which was opened and curetted, and the superficial portion removed for section. Dr. Bull reported: "Sections show much foreign body giant cell formation, areas of polymorph accumulation and areas of squamous epithelial cells. The picture suggests a deposit of a squamous cell epithelioma. There is no evidence of a tuberculous inflammation."

A course of deep X ray therapy was given in April, 1933, and the patient has remained well since then, except for some vague thickening at the site of the original tumour. Clinically this may be no more than scar tissue.

CASE VI (registered number 2198).—T.N., a male, aged sixty-six years, noticed a small lump on the right side of his neck in May, 1933. This grew slowly until September, when he first noticed pain in the right side of his head. His voice became a little husky. In October there was a firm tumour with well-defined edges high up on the right side of the neck beneath the sterno-mastoid muscle, which it was infiltrating. It was fixed to the carotid artery and other structures and was excised on October 21, together with portion of the sterno-mastoid muscle, the jugular vein and adjacent structures. Examination of the mouth, nose, and throat did not reveal any primary tumour. Dr. McLaughlin reported: "Sections show a squamous cell epithelioma."

Deep X ray therapy was given in November, 1933, but in February, 1934, there was a hard tumour fixed in the right submaxillary region, extending forwards to involve the right half of the submental region. Further deep X ray treatment is in progress.

CASE VII (registered number 2291).—J.T.R., a male, aged fifty-one years, noticed a swelling in the anterior part of the neck in August, 1933. It improved with hot foments, but became larger and harder on the right side. Some lymph glands were removed from the right side of the neck on November 4, 1933, but showed no abnormality. The lump persisted, and a further incision was made on December 27, 1933, when a widely infiltrating tumour was found, very hard in some parts and much softer in others. It was present beneath the upper part of the right sterno-mastoid muscle. Careful visual examination and palpation under a general anæsthetic did not reveal a primary growth in the mouth or pharynx, and radiographs excluded a primary growth in the lungs. The Wassermann test yielded no reaction. Dr. Bull reported: "Sections showed a squamous cell carcinoma with much round cell reaction surrounding the epithelial cell masses." There were no cell nests.

Deep X ray therapy was given in January, 1934, followed by improvement, but the vague thickening that remains is almost certainly a residuum.

Group II: Squamous Epitheliomata with Cell Nests, Under the Jaw or Near the Mid-Line, Probably not Arising from Branchial Epithelium.

Group II comprises squamous epitheliomata with cell nests, the growths occurring under the jaw or near the mid-line. Their origin is probably not from the branchial epithelium. Two cases (Cases VIII and IX), both in males, aged 59 and 70 years, fall into this group. In both the tumour occupied the submental region.

CASE VIII (registered number 595).—H.Y.S., a male, aged fifty-nine years, noticed a tumour in the anterior mid-line of the neck in June, 1930, with some dysphagia, which was relieved following operation on August 7. Incision revealed a hard, rounded tumour, about three centimetres in diameter, infiltrating the superficial muscles between the mandible and the hyoid bone. The floor of the mouth was not involved, nor could a primary growth be demonstrated elsewhere in the mouth or pharynx. There were no palpable lymph glands in the neck. Dr. Bull reported: "Sections show a squamous cell epithelioma. It may have arisen from the lingual duct (from which dermoids occasionally develop) or from some cellular remnants of the duct."

One month later a recurrent tumour developed to the left of the scar and a radium pack was applied on October 3. Resolution of the tumour followed, but subsequently he developed metastases to glands in both upper deep cervical groups, and these were not obviously affected by a further radium pack. He died on May 4, 1931.

CASE IX (registered number 1531).—J.L., a male, aged seventy years, noticed a hard lump under the right side of the mandible in December, 1931; this remained unaltered for about seven months, but then increased in size fairly rapidly. On admission in August, 1932, there was a hard, fixed tumour of vague outline situated in the submental region; the skin over it was red and shiny. Leucoplakia of the lower lip was noticed, but there was no clinical evidence of a neoplasm within the buccal cavity. A biopsy was performed and Dr. Bull reported: "Sections show a squamous cell epithelioma." There was very intense cornification with cell nests, but normal skin on the surface.

Deep therapy was given in August and October, 1932, but the progress of the lesion was not affected; the tumour eventually ulcerated, there was much pain, and death occurred on May 15, 1933.

Group III: Epitheliomatous Tumours with Cells Presenting Transitional Appearance or Suggesting a Basal Cell Type: Origin Probably from Branchial Epithelium.

Group III comprises epitheliomatous tumours, with the cells presenting a transitional appearance or suggesting a basal cell type. Their origin seems best explained as from the branchial epithelium. Three cases (X, XI, XII), all occurring in males, aged 57, 66, 70 years, belonged to this group. In all the tumour was mesial to and anterior to the upper portion of the sterno-mastoid muscle.

CASE X (registered number 1299).—P.M.O'H., a male, aged seventy years, noticed a painless swelling on the left side of the neck about September, 1931. This increased in size shortly before his admission to hospital in March, 1932, when there was a fixed mass of glands beneath the upper third of the left sterno-mastoid muscle. A primary tumour could not be demonstrated in the mouth or pharynx. A biopsy was performed and Dr. Bull reported: "Sections show a deposit of a carcinoma. The tumour cell is probably from a squamous or transitional epithelium, but there is no cornification."

Deep X ray therapy was commenced in April, 1932, but the patient declined to complete the course prescribed. There was improvement in the tumour before his death in August, 1932.

CASE XI (registered number 1325).—J.C.S., a male, aged sixty-six years, noticed a lump on the left side of his neck in February, 1932, with pain up the left side of his head. One month later some teeth were extracted, and this was followed by relief of pain, although the tumour in the neck became larger. In April, 1932, there was a hard, rounded mass, five centimetres in diameter, behind and below the left angle of the mandible, infiltrating the upper third of the left sterno-mastoid muscle. A primary tumour could not be found within the mouth or pharynx. The Wassermann test yielded no reaction. Dr. Bull reported on a biopsy specimen: "Sections show a carcinoma. It is not of the acanthoma type, but more of the basal cell type."

Deep therapy was commenced in April and caused a brisk epidermolysis. In June vague thickening marked the site of the original tumour. He died at home in September, 1932, of "chronic myocarditis".

CASE XII (registered number 1636).—S.K., a male, aged fifty-seven years, noticed a painless lump in the right side of his neck in July, 1932. Soreness of his throat on swallowing developed at the end of September, 1932. In October, 1932, there was a fixed tumour in the right side of the neck, extending from just below the tip of the mastoid process down to the level of the cricoid cartilage. Incision showed that it was firmly fixed to the jugular vein and surrounding structures and was inoperable. Dr. Bull reported: "Sections show a carcinoma. The tumour is not a frankly squamous cell epithelioma, but is of the transitional cell type. The mass does not appear to be associated with a lymphatic gland. It may have arisen in a branchial rest or be a deposit from a tumour of the mouth or naso-pharynx."

A primary growth could not be found in the mouth, pharynx, or lungs. Deep X ray therapy was commenced in October, 1932, but at the completion of approximately one-third of the prescribed dose the patient complained of the general effect of the treatment and declined to continue. Subsequently he went to the New South Wales Home for Incurables, where, in April, 1933, radium treatment was applied to the right side of his neck, approximately 14,600 milligramme-hours with buried tubes and needles being given. At that time discrete glands were palpable below the original tumour. He returned to us in June, 1933, with much induration of the upper right half of his neck and an ulcer of the surface, but the tumour was smaller. Complaint was made of much pain, and in September 1933, a large gland was palpable above the inner end of the right clavicle. He died from cachexia in December, 1933.

Group IV: Epithelioma Suggesting an Origin from a Nasal Sinus.

Group IV comprises epithelioma suggestive of an origin from a nasal sinus. One case (XIII), occurring in a male, aged sixty-five, was in this group. The site of origin was indefinite.

CASE XIII (registered number 2036).—A.W., a male, aged sixty-five years, noticed a lump in the right side of the neck in February, 1933. This increased in size and ulcerated in June, 1933. He had a slight cough, but no symptoms suggestive of trouble in the nose. On his admission to hospital in July, 1933, there was extensive ulceration of the right side of the neck extending across the mid-line anteriorly, with enlarged matted glands on the left side. The circumference of the neck was much increased and it was impossible to determine with any accuracy the site of origin. Portion was removed for section and Dr. F. R. Hone reported: "Section shows secondary deposits of a carcinoma. It is impossible to say from the section the origin of the primary growth. It is not a squamous epithelioma. It might possibly originate in the nasal sinuses."

Clinical and radiographic examination failed to reveal a primary tumour in the mouth, nasal accessory sinuses, mediastinum or lungs. Deep therapy in August, 1933, was followed by some improvement in the ulceration, but the latter did not heal. He died in November, 1933.

Group V: Squamous Epithelioma Secondary to Growth Surrounding the Pituitary Body: Origin from Epithelium of Rathke's Pouch.

Group V comprises a squamous epithelioma secondary to a growth surrounding and destroying the pituitary body. The origin was from the epithelium of Rathke's pouch. This occurred in Case XIV, the patient being a male, aged fifty. The scar of excision was situated in the upper part of the neck laterally.

CASE XIV (registered number 719).—H.J.R.G., a male, aged fifty years, was admitted to hospital in January, 1931, when he gave a history of having had a lump excised from the right side of the neck in 1929 in a Sydney hospital. In May, 1930, a radium pack was applied in the Melbourne Hospital to the affected area, and evidence of delayed healing of the skin was still present when he was admitted to the Adelaide Hospital. There was thickening of the tissues at this site, but no obvious tumour up to the time of his death, which occurred on March 24, 1931.

At autopsy (number 46/31) there was found a squamous cell epithelioma, without cell nests, replacing the pituitary gland and extending as a solid mass downwards, connecting up with dense fibrosis in the upper part of the neck, thus extending to the irradiated area. The fibrous tissue contained deposits of the newgrowth, which was thought to have arisen in remnants of Rathke's pouch.

Group VI: Diffuse Carcinomatous Growth, Possibly Epitheliomatous: Origin Unknown.

Group VI comprised a diffuse carcinomatous growth, possibly of epitheliomatous origin. The source was unknown, though the branchial epithelium cannot be excluded. Dr. Bull thought it might be endotheliomatous. This occurred in a male, aged forty-eight (Case XV). The tumour was situated behind and below the angle of the mandible.

CASE XV (registered number 1146).—E.H., a male, aged forty-eight years, noticed a small tumour behind the left angle of the mandible in May, 1931. This increased in size steadily, and one year later there was a group of enlarged glands palpable in that area. One was removed for section and Dr. Bull reported: "Sections show a deposit of a

malignant newgrowth, apparently a deposit of a large spheroidal cell carcinoma. It is possible, however, that it is an endothelioma." (There are some large and very irregular cells, so that diffuse infiltration by squamous epitheliomatous cells cannot be excluded.) A primary growth could not be found within the mouth or pharynx, while a radiograph of the lungs showed a normal picture.

Deep X ray therapy was given in May, 1932, followed by local improvement, which was maintained until February, 1933. At that time the patient complained of a cough, and a radiograph shortly afterwards showed a circumscribed mass projecting into the right lung root. The chest was treated with deep X rays and there was some further temporary improvement. He died in September, 1933, from the effects of his intrathoracic tumour.

Group VII: Indefinite Neoplasm.

Group VII comprises an indefinite neoplasm, possibly an endothelioma, possibly not a newgrowth at all. This occurred (Case XVI) in a male, aged seventy years.

CASE XVI (registered number 608).—W.G.H., a male, aged seventy years, noticed a small lump in the left side of his neck in May, 1930. This was painless at first, but became painful with increase in size. In September, 1930, there was a cystic tumour beneath the upper part of the left sterno-mastoid muscle. A portion was excised at the end of September and Dr. Bull reported: "Sections show a fibrous wall infiltrated by polymorphs, lymphocytes and plasma cells. There are groups of cells present with indefinite outlines which resemble neoplastic cells, possibly endothelial in origin. The portion is too small for a more definite opinion."

When deep X ray therapy was commenced in October, there was a tumour about six centimetres in diameter on the left side of the neck, with a reddish surface, of even consistency, and attached to surrounding structures. Subsequent to deep X ray treatment the tumour became smaller and was replaced by a thickening of the sterno-mastoid muscle. Glands were noticed in relation to the carotid vessels, and in January, 1931, radium was applied on a two-centimetre wax "collar", followed by a mild degree of epidermolysis. The glands became smaller, but in June, 1931, he developed epigastric tenderness and pain one hour after meals. There was an enlarged liver and the stomach was pushed to the left, probably by glands along the lesser curvature. There was no free hydrochloric acid in the stomach and lactic acid was present, while occult blood was detected in the stools. He died in February, 1932. A *post mortem* examination was not allowed, but it was considered that death was due to carcinoma of the stomach; the cystic nature of the lump in the neck, together with the histological appearance of the growth, suggested that this was not secondary to the gastric tumour, but that it was a different lesion. A thickening in the neck was still present at the time of death.

Summary.

1. The possible sources of cryptogenic carcinomata of the neck are considered.

2. The arguments in support of an origin from remnants of the branchial epithelium are set out, and to such an origin we attribute some, probably the majority, of such growths.

3. Some of the cases submitted have been so thoroughly examined and have been so long under observation that a primary growth in a neighbouring structure could hardly have escaped recognition. Even though such a possibility be granted, the cases submitted seem too many to be all thus explained.

4. An origin from displaced surface epithelial cells can, if it ever happens, be considered only as a freak occurrence.

5. All the cases occurred in males over forty-nine years of age. One was bearded, the others shaved. It can hardly be seriously suggested that accidents in shaving, as under 4, can have been a factor, but the vigorous growth of the hair follicles of the male might be a reason for undue activity eventually manifesting itself in adjacent dormant groups of branchial epithelial cells.

6. The growths seem mostly to have an origin under the upper part of the sterno-mastoid muscle.

7. Treatment was invariably unavailing, though perhaps some mitigation of suffering has resulted.

8. Details of the deep X ray technique are stated.

9. A *résumé* of sixteen case histories is set out.

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THE VENOMS OF THE BROAD-HEADED SNAKE (HOPLOCEPHALUS BUNGAROIDES) AND OF THE YELLOW-BANDED SNAKE (HOPLOCEPHALUS STEPHENSI).

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THE three species of the genus *Hoplocephalus*, *Hoplocephalus bitorquatus* Jan, *Hoplocephalus stephensi* Krefft, and *Hoplocephalus bungaroides* Boie, are all relatively uncommon. The first two occur patchily distributed in Queensland and New South Wales, and the third is restricted to the coastal areas and ranges of New South Wales.

According to Kinghorn,(1) *Hoplocephalus bitorquatus* grows to a length of a little over 50 centimetres (20 inches) and is venomous, but not deadly. We have not had a living specimen of this species.

Hoplocephalus stephensi is somewhat larger, attaining a length of 75 centimetres (30 inches). We obtained a single living specimen captured by Mr. Tom Eades at Brigalow in south-western Queensland. It is a vigorous and aggressive snake and, despite its small size, our observations of its venom would tempt us to modify Mr. Kinghorn's statement that "it is venomous and a bite would cause pain and swelling, but it is not deadly". We think it likely that a bite by a full grown specimen would cause severe general symptoms.

Hoplocephalus bungaroides grows to a much larger size, according to Kinghorn to over 150 centimetres (five feet). It is said to be an extremely aggressive snake and must be regarded as dangerous to man and the larger animals. We have not been able to obtain a living specimen of this species, but Professor H. G. Chapman kindly gave me

fifty milligrammes of the dry venom. This was the total of four successive milkings from a single snake, which was in his possession in 1911. The average venom yield of this specimen was therefore about twelve milligrammes of dry venom. The venom was in first rate condition, having been kept *in vacuo* since its collection, and was easily and completely soluble in saline. It was used in making the following observations.

The Venom of Hoplocephalus Bungaroides.

The Results of Intravenous Injection in Wild Rabbits.

The venom contains a powerful thrombin which is unstable in the dilute solutions necessary for testing its action. It is very difficult, therefore, to estimate accurately its potency *in vivo*. In three successive series of experiments the diluted venom (0·01 milligramme per cubic centimetre) was in the earliest injections more effective than in the same or higher dosage after an hour at laboratory temperature (18° to 19° C.).

For instance, on the first occasion of testing, wild rabbits weighing 1·52, 1·12, 1·48, 1·29 and 1·61 kilograms received the first, 0·0025, and the remainder 0·002 milligramme per kilogram injected into a marginal ear vein. The first three animals died in five, five and a half, and four and a half minutes with whipped-out fibrin in the right heart, and the last two survived without symptoms.

This might have been regarded as indicating variation of individual resistance, but at each test similar results were obtained. The results of the final testing are set out in Table I. In estimating the lethal dose of venoms containing thrombin by intravenous injection, the size of the total dose seems to be more important than the dose estimated per kilogram of body weight. Generally speaking, larger animals injected with the same dose per kilogram die more quickly with more extensive thrombosis. For this reason I have given the total doses in Table I. The rabbits used had not been fed for twelve hours.

TABLE I.

Results of intravenous injection of the venom of Hoplocephalus bungaroides *in wild rabbits.*

Time of Injection.	Weight in Kilogrammes.	Dose in Milligramme per Kilogram.	Total Dose in Milligramme.	Result.
p.m.				
4·0	1·17	0·004	0·0047	Death in 3½ minutes.
4·6	1·31	0·003	0·0039	Death in 4 minutes.
4·12	1·33	0·002	0·0027	Death in 5½ minutes.
4·25	1·09	0·002	0·0022	Survived without symptoms.
4·40	1·26	0·003	0·0038	Death in 5 minutes.
4·50	1·15	0·003	0·0035	Death in 8 minutes.
5·5	1·48	0·003	0·0044	Severe symptoms 10 to 15 minutes after injection. Survived.

The thrombin in this venom is of the same order of potency as that of the tiger snake, *Notechis scutatus*, and brown snake, *Demansia textilis*, and, if it were tested freshly made up by injecting a large number of animals simultaneously, the certainly lethal dose would probably prove to be about 0·002 milligramme per kilogram in wild rabbits. That this dose is near the limit is indicated by the fact that, in those rabbits which died, clotting was found only in the right heart and not in the large vessels.

Miss Williams has made a few observations on the coagulating power of this venom *in vitro* on the citrated plasmas of man and of the rabbit and guinea-pig. The citrated plasmas were obtained by bleeding into an equal amount of isotonic sodium citrate solution. For the tests 0·2 cubic centimetre of citrated plasma was added to 0·1 cubic centimetre of saline solution containing various doses of venom. In two series of observations the stock venom solution (1·0 milligramme per cubic centimetre) was used freshly made up and after standing at laboratory temperature (18° C.) for four hours. The saline solution in which the venom was made up both for these tests and for tests *in vivo* had a pH of 7·0.

In Table II are set out the results of these two parallel experiments. The coagulation times are in minutes, except where stated to be hours.

TABLE II.

The coagulation times of citrated plasmas of man, rabbit and guinea-pig by Hoplocephalus bungaroides *venom.*

Dose of Venom in Milligramme.	Coagulation Times in Minutes.					
	Guinea-pig Plasma.		Rabbit Plasma.		Human Plasma.	
	Fresh.	Four Hours Old.	Fresh.	Four Hours Old.	Fresh.	Four Hours Old.
0·1	1½	1½	1	2	1½	4
0·01	2	2½	1½	2⅝	2½	4½
0·001	3½	N	3½	13	5½	N
0·0001	13	N	12	1¼ hours	13½	N
0·00001	2¼ hours	N	2¼ hours	N	1 hour	N
0·000001	N	N	N	N	N	N

N = No coagulation.

The impairment of coagulating power on standing in dilute solution is more strikingly shown with human and guinea-pig plasmas than with that of the rabbit. It may be recalled that the thrombin of the venom of the brown snake, *Demansia textilis*, is also unstable in dilute solution on the acid side of neutrality (Kellaway[2]), and it is possible that uniform results with the venom of *Hoplocephalus bungaroides* might have been obtained in buffered solution.

The Effect of Subcutaneous Injection in Wild Rabbits.

The toxic action of the venom, excluding the effect of the thrombin, was estimated by subcutaneous injection in rabbits, guinea-pigs and rats. In wild rabbits it proved to be much less toxic than the venom of the tiger snake, *Notechis scutatus*, and of about the same order as that of the copperhead, *Denisonia superba*. The results of a few experiments are set out in Table III. The venom was injected subcutaneously in the abdominal wall in a concentration of 1·0 milligramme per cubic centimetre.

TABLE III.

Results of subcutaneous injection of the venom of Hoplocephalus bungaroides *in wild rabbits.*

Average Weight in Kilograms.	Number of Animals.	Dose in Milligramme per Kilogram.	Results.
1·3	4	0·7	One died in less than 20 hours, one in 24 hours, one in 47 hours, and one survived.
1·1	5	0·5	One died in less than 46 hours, one in 48 hours, three had severe symptoms, but survived.
1·55	1	0·4	Died in less than 46 hours.
1·23	4	0·3	All had slight symptoms—anorexia, and local discomfort, but survived.

The certainly lethal dose is evidently rather more than 0·7 milligramme per kilogram. The venom has a powerful local effect, causing extensive hæmorrhagic œdema spreading from the region of the injection. With doses of this order the rabbits go off their feed and look sick after about twelve hours. During the last few hours before death they exhibit symptoms due to the action of the venom on the peripheral nervous system, ataxia and paresis of the limbs. There is some dyspnœa generally associated with extensive lesions in the lungs at autopsy. Death takes place from respiratory failure.

Post mortem the blood is fluid and the coagulation time delayed. Blood drawn from the heart immediately after death clots in twenty-five minutes or more instead of in from five to eight minutes, as in normal rabbits. Stimulation of the phrenic nerves shows some paralysis of the endings in the diaphragm, though this is not very striking with these doses. In one rabbit, for example, which died after forty-seven hours with extensive hæmorrhagic consolidation of the anterior and upper parts of both lungs, faradic stimulation of the left phrenic nerve gave feeble contraction of the diaphragm with the coils separated sixteen centimetres, whereas the diaphragm stimulated directly contracted with the coils separated twenty to twenty-two centimetres.

In order to ascertain what the action of the venom is, apart from the production of serious visceral lesions, much larger doses were given to rabbits under anæsthesia. Two animals died shortly after the injection of three milligrammes and two milligrammes per kilogram subcutaneously, and in each whipped-out fibrin was found in the right side of the heart. It was necessary therefore to heat the venom and destroy the thrombin to ascertain what was the behaviour of the remaining constituent or constituents of the venom. The venom in a concentration of one milligramme per cubic centimetre in saline solution was placed in a boiling water bath for three minutes and was used in the following experiment:

EXPERIMENT 1.—A wild rabbit weighing 1·4 kilograms was used.

At 12.52 p.m. 0·7 cubic centimetre of "Numal" was given intravenously.

At 1 to 1.30 p.m., under light ether anæsthesia, tracheal and carotid cannulas were tied in and the upper root of the right phrenic nerve was exposed in the neck. The diaphragm contracted strongly to faradic stimulation of the phrenic nerve with the primary and secondary coils separated 50 centimetres.

At 1.45 p.m. four milligrammes of heated *Hoplocephalus bungaroides* venom were injected subcutaneously.

At 1.50 p.m. the blood pressure was 102 millimetres of mercury.

At 2.5 p.m. the blood pressure was 82 millimetres; the respirations numbered 32 per minute.

At 2.45 p.m. the blood pressure was 96 millimetres; respirations numbered 37 per minute. The diaphragm responded well with phrenic stimulation at 36 centimetres separation.

At 3.4 p.m. the blood pressure was 108 millimetres; Traube Hering waves were noted and strong dyspnœa was present.

At 3.10 p.m. the blood pressure tracing showed strong vagal slowing of the heart. The blood was dark. Respirations numbered 24 per minute, they were wholly costal. No diaphragmatic response to faradic stimulation of the phrenic nerve occurred at five centimetres separation of the coils.

At 3.19 p.m. convulsions were noted.

At 3.20 p.m. artificial respiration was started. The systolic blood pressure fell to 88 millimetres.

At 3.40 p.m. the blood pressure was 48 millimetres; there was a striking diminution of cardiac output.

At 3.54 p.m. sudden death from circulatory failure occurred.

Post mortem there were no local lesion and no obvious lesions in the lungs nor thrombi in the pulmonary vessels. The right side of the heart was distended, but contained no whipped-out fibrin and the blood from the heart clotted in nine minutes.

This experiment exhibits several points of interest. In the first place, at autopsy there was no sign of local œdema or hæmorrhage round the site of injection. It is therefore likely that that part of the venom complex which causes this effect is, like the thrombin, thermolabile. The dose used in this experiment caused complete paralysis of the motor endings in the diaphragm within one and a half hours, but was so large as to cause death from circulatory failure in a little over two hours. There were no extensive lung lesions to account for this, and it appeared likely that the venom in this concentration either caused peripheral circulatory failure or acted directly on the heart itself.

The Results of Subcutaneous Injection in Guinea-Pigs.

All the guinea-pigs used were of average weight 300 grammes, varying between 275 and 330 grammes. The first two groups in Table IV were injected with venom in a concentration of one milligramme per cubic centimetre, the last three in a concentration of 0·1 milligramme of venom per cubic centimetre.

TABLE IV.

The results of the subcutaneous injection of the venom of Hoplocephalus bungaroides *in guinea-pigs.*

Average Weight in Grammes.	Number of Animals.	Dose in Milligramme per 100 Grammes.	Results.
300	2	0·08	Died in 2 hours 48 minutes and in 4 hours 8 minutes.
315	2	0·06	Died in 2 hours 7 minutes and in 2¼ hours.
300	6	0·04	One died in 6¼ hours, two in less than 14 hours, and three in less than 22 hours.
282	4	0·03	Three died in 11½ hours and one in 20 hours.
300	2	0·02	One died on the third day and one survived without symptoms.

The certainly lethal dose is in the neighbourhood of 0·3 milligramme per kilogram. The symptoms are not distinguishable from those following the injection of other potent Australian snake venoms. The animals which received 0·04 milligramme per kilogram exhibited dyspnœa within four hours and in seven hours were collapsed and paralysed in the hind limbs. They all had difficulty in respiration, with excessive secretion from the mouth and upper respiratory passages. There were "starting movements" from time to time. In all those in which death was observed, it took place by failure of respiration, the heart continuing to beat after respiration had ceased. The blood was fluid and incoagulable or exhibited delayed coagulation. In several animals stimulation of the phrenic nerve and of the diaphragm immediately after death showed that there was a pronounced peripheral action on the nerve endings, much stronger stimuli being required to cause contraction of the diaphragm indirectly when the phrenic nerve was stimulated than when the muscle was stimulated directly. *Post mortem* all the animals had hæmorrhagic œdema at the site of injection. The tissues were cyanosed, the lungs were congested with numerous petechial hæmorrhages. In one or two animals the small intestines and adrenals were also slightly congested.

The Results of Subcutaneous Injection in Rats.

The rats used were black and white and albino, and the venom was injected in a concentration of 0·1 milligramme per cubic centimetre. The results are set out in Table V.

TABLE V.
The results of subcutaneous injection of the venom of Hoplocephalus bungaroides *in rats.*

Average Weight in Grammes.	Number of Animals.	Dose in Milligramme per 100 Grammes.	Results.
231	2	0·5	Died in 1 hour 18 minutes and in 1 hour 46 minutes.
245	2	0·3	Died in 1 hour 38 minutes and in 1 hour 42 minutes.
240	2	0·2	Both died in 1 hour 20 minutes.
238	8	0·1	One died in 2 hours 43 minutes, one in 2 hours 55 minutes, one in 3 hours 10 minutes, one in 3 hours 55 minutes, one in 4 hours 50 minutes, one in 4 hours 57 minutes, one in less than 22 hours, and one survived.
233	6	0·08	One died in 3 hours 10 minutes, one in 5 hours. The remainder had extremely severe symptom, for from 12 to 36 hours, but survived.
234	6	0·07	One died in 2½ hours, one in 4½ hours, and the remainder survived after severe symptoms lasting from 12 to 36 hours.
209	4	0·05	All survived without symptoms.

The certainly lethal dose is about 0·1 milligramme per 100 grammes, or 1·0 milligramme per kilogram. The toxicity of the venom for this species is therefore well inside the range of activity of the most toxic Australian snake venoms (death adder 0·4, tiger snake more than 0·4, and copperhead 2·4 milligrammes per kilogram).

The symptoms exhibited were very uniform with doses in the neighbourhood of the certainly lethal dose. Within three or four hours all the animals were severely affected with dyspnœa, wet mouths and paralysis, more or less extensive, starting in the hind limbs. Later, they became collapsed, with continued dyspnœa and starting movements. Death took place with failure of respiration. *Post mortem* the blood was fluid and incoagulable or clotted only after much delay. There was hæmorrhagic œdema at the site of the injection. The tissues were cyanosed. The lungs were congested and exhibited numerous hæmorrhages. A few rats tested showed some degree of curari-like paralysis of the phrenic nerve endings in the diaphragm. All the rats exhibited hæmoglobinuria as an early symptom, and the kidneys at autopsy were dark with altered blood.

The Hæmolytic Power of the Venom of Hoplocephalus Bungaroides.

The hæmolytic power of the venom of *Hoplocephalus bungaroides* is extremely feeble. In rats it caused hæmoglobinuria, but this symptom was not observed in rabbits and guinea-pigs. Miss F. E. Williams has tested the hæmolytic power of the venom *in vitro* upon the washed red blood corpuscles of man and of the rabbit and guinea-pig in 3% suspension in saline solution and upon defibrinated blood diluted with saline solution to an equivalent degree. The hæmolytic system had a volume of 0·3 cubic centimetre—0·1 cubic centimetre of 3% corpuscles and 0·2 cubic centimetre of saline containing 0·1 milligramme, 0·01 milligramme and 0·001 milligramme of venom. Washed guinea-pig cells were completely hæmolysed in two and a half hours by 0·1 milligramme. In eighteen hours washed human red blood cells were completely hæmolysed by the same dose of venom, and washed rabbit cells showed a trace of hæmolysis. Defibrinated guinea-pig and human blood were not hæmolysed even by the largest dose, and defibrinated rabbit blood with 0·1 milligramme of venom exhibited only a trace of hæmolysis after eighteen hours.

Comparison of the Toxicity of this Venom with that of Common Australian Venomous Snakes.

The lethal doses of this venom by subcutaneous injection in the wild rabbit, guinea-pig and rat are more than 0·7, 0·3 and 1·0 milligramme per kilogram. For the tiger snake, *Notechis scutatus*, the lethal doses are 0·045, 0·02 and more than 0·4 milligramme per kilogram; for the death adder, *Acanthophis antarcticus*, 0·15, 0·15 and 0·4 milligramme per kilogram; for the copperhead, *Denisonia superba*, 0·7, 0·06 and 2·4 milligrammes per kilogram; and for the brown snake, *Demansia textilis*, more than 0·2, 0·025 and 0·6 milligramme per kilogram. The venom, judged by injection in these three species of animals, is of about the same order of toxicity as that of the copperhead.

The venom resembles that of the tiger snake and brown snake in possessing a powerful thrombin and in being only feebly hæmolytic. It causes more

extensive local lesions than any of these other venoms, though this is partly accounted for by the larger doses used.

The Neutralization of the Venom of Hoplocephalus Bungaroides by Tiger Snake and Copperhead Antivenenes.

A snake which reaches the length of over 150 centimetres (five feet) and which is very broad-headed, probably yields considerable quantities of venom at a bite, though we have no certain information upon this point. In view of the rarity of this species it is unlikely that specific antivenene will ever be prepared against its venom. It is important, therefore, to ascertain what neutralizing power the available monovalent antivenenes (tiger snake and copperhead) possess against it.

The two antivenenes used were prepared by Dr. F. G. Morgan at the Commonwealth Serum Laboratories. The venom and antivenene were mixed and allowed to stand for an hour at laboratory temperature (19° C.) before being injected. The results of neutralization experiments are set out in Table VI.

TABLE VI.

Neutralisation of the venom of Hoplocephalus bungaroides *by monovalent tiger snake and copperhead antivenenes.*

Weight of Guinea-pig. Grammes.	Mixture Injected.	Results.
298	Tiger snake antivenene 1·0 cubic centimetre + 0·1 milligramme *Hoplocephalus* venom.	Survived without symptoms.
306	Tiger snake antivenene 0·5 cubic centimetre + 0·2 milligramme *Hoplocephalus* venom.	Survived without symptoms.
288	Tiger snake antivenene 0·5 cubic centimetre + 0·25 milligramme *Hoplocephalus* venom.	Died in 18 hours.
272	Tiger snake antivenene 0·5 cubic centimetre + 0·3 milligramme *Hoplocephalus* venom.	Died in 5 hours.
284	Copperhead antivenene 1·0 cubic centimetre + 0·1 milligramme *Hoplocephalus* venom.	Survived without symptoms.
285	Copperhead antivenene 0·5 cubic centimetre + 0·2 milligramme *Hoplocephalus* venom.	Died in 28 hours.
270	Copperhead antivenene 0·5 cubic centimetre + 0·3 milligramme *Hoplocephalus* venom.	Died in 9 hours.

Photograph from life of *Hoplocephalus stephensi*, by Mr. Donald Thomson.

Tiger snake antivenene which neutralized 0·29 milligramme of tiger snake venom, neutralizes between 0·24 and 0·33 milligramme of *Hoplocephalus bungaroides* venom per cubic centimetre. Copperhead antivenene, which neutralized 0·15 milligramme of copperhead venom, neutralizes less than 0·22 milligramme of *Hoplocephalus bungaroides* venom per cubic centimetre.

The use of monovalent tiger snake antivenene is indicated for the treatment of bites by *Hoplocephalus bungaroides*, since 100 cubic centimetres of antivenene of this potency can neutralize at least 24 milligrammes of the venom.

The antivenene also neutralizes the thrombin of the venom of *Hoplocephalus bungaroides*.

A wild rabbit weighing 1·23 kilograms was injected intravenously without any effect with a mixture of 1·0 cubic centimetre of tiger snake antivenene and 0·2 milligramme of *Hoplocephalus bungaroides* venom. This dose of venom is about one hundred lethal intravenous doses. The mixture was allowed to stand an hour at 19° C. before injection and part of the destruction of thrombin is doubtless due to its natural instability. Even allowing for this, the antivenene had neutralized many certainly lethal intravenous doses of venom.

The Venom of Hoplocephalus Stephensi.

Our single specimen of *Hoplocephalus stephensi* was captured in 1930 and remained in good condition for three months in captivity. For the excellent photograph taken during life (Figure I) and the following description I am indebted to Mr. Donald Thomson.

Length 698·0 millimetres, tail 106·0 millimetres, abdominals 216, anal undivided, subcaudals 57 all single, body scales in 21 rows, transverse body scales at every twentieth abdominal.

20th	40th	60th	80th	100th	120th	140th	160th	180th	200th
21	21	21	21	21	21	19	17	17	15

General Description. Head very distinct from body, particularly when snake is agitated; broad and much flattened. Angles of jaws prominent when annoyed. Eye small, with circular pupil, its length greater than its distance from mouth. Abdominal scales with keel on each side; body scales smooth. Rostral only just visible from above and broader than long (4·5 millimetres by 3·5 millimetres). Frontal (6·5 millimetres by 4·5 millimetres) longer than its distance from the snout and approximately one and a half times as long as broad; nearly twice as broad as the supraocular (2·5 millimetres) and a little more than two-thirds as long as the parietals (9·0 millimetres). Nasal divided, and in contact with the single preocular, 2 postoculars, temporals 2 + 2, lower anterior so deeply wedged between 5th and 6th supralabials as almost to touch upper lip. Six supralabials, 3rd and 4th entering eye. First three infralabials only in contact with anterior chin shields.

Colour. Black, with about 35 regularly spaced bands of light ash brown on body, bands extending to, but becoming less clearly defined on tail. Head dull greyish brown marbled with black. Some of the supra- and infralabials and temporals bear large white markings. The chin and throat are creamy white, marked with blackish brown. Ventral surface brownish marbled black with a line of creamy white marks on anterior end of abdomen.

The primary venom yield on February 20, 1930, was 5·8 milligrammes; at successive monthly milkings it yielded 4·1, 2·8, and on May 30, after three months in captivity, 2·0 milligrammes of venom. The venom was very similar, both qualitatively and quantitatively, to that of *Hoplocephalus bungaroides*.

Effects of Intravenous Injection in Domestic Rabbits.

The venom was injected in a concentration of 0·01 milligramme per cubic centimetre into a marginal ear vein. The results are set out in Table VII. In the first two animals the venom was injected in a concentration of 0·1 milligramme per cubic centimetre, and in the remainder in a concentration of 0·01 milligramme per cubic centimetre.

TABLE VII.

Results of intravenous injection of the venom of Hoplocephalus stephensi *into rabbits.*

Number of Animals.	Weight or Average Weight in Kilograms.	Dose in Milligramme per Kilogram.	Results.
1	1·66	0·1	Died in 1½ minutes.
1	1·1	0·01	Died in 3½ minutes.
1	1·05	0·005	Died in 3 minutes.
4	1·95	0·003	Died in 3, 4, 4½ and in 7 minutes.
2	1·83	0·0025	Died in 6 and in 12 minutes.
2	2·31	0·002	Both survived without symptoms.

The certainly lethal dose is about 0·0025 milligramme to 0·003 milligramme per kilogram. The venom, like that of *Hoplocephalus bungaroides*, is powerfully coagulant in action. The symptoms produced were those typical of intravascular thrombosis. In those animals which received 0·0025 milligramme per kilogram, whipped-out fibrin was found in the right side of the heart, but with larger doses there was *ante mortem* clotting in the pulmonary and portal veins and in the inferior *vena cava*. The further study of the toxicity of the venom was made by subcutaneous injection in rabbits and guinea-pigs.

The Results of Subcutaneous Injection in Rabbits.

The venom was injected subcutaneously in the abdominal wall into three domestic rabbits in a concentration of 1·0 milligramme per cubic centimetre. Two rabbits, weighing 2·13 and 2·27 kilograms, received 0·25 and 0·1 milligramme per kilogram respectively, and survived without symptoms. A third received 1·0 milligramme per kilogram and died in three and a half hours. It was obviously sick after two hours and rapidly became feeble and collapsed. There was no definite paralysis. Death occurred by respiratory failure. There was intense hæmorrhagic extravasation over the whole abdominal wall, but no hæmoglobinuria or other evidence of hæmolysis. The lungs were flabby and wet, but there were no other gross changes in the organs. The blood was fluid in the heart and vessels and did not clot *in vitro* in one hour.

A further study of the action of the venom was made by injecting 2·0 milligrammes of the venom (heated in 0·1% solution for three minutes at 100° C.) subcutaneously in a wild rabbit under "Numal" anæsthesia.

The upper phrenic root was exposed in the neck and carotid and tracheal cannulas were inserted. Unfortunately the dose did not cause complete paralysis of the phrenic endings in six hours and the animal was still in good condition when the experiment had to be abandoned. After three hours and until the end of the experiment there was evidence of partial paralysis of the phrenic motor endings. At the beginning of the experiment the diaphragm contracted strongly when the phrenic nerve was stimulated with the coils separated 50 centimetres, whereas by the beginning of the fourth hour till the end of the sixth only a feeble response could be obtained with the coils separated 30 to 23 centimetres.

The Results of Subcutaneous Injection in Guinea-Pigs.

The results of a few observations in guinea-pigs are set out in Table VIII. The venom was injected subcutaneously into the abdominal wall in a concentration of 0·1 milligramme per cubic centimetre.

TABLE VIII.

The effect of subcutaneous injection of the venom of Hoplocephalus stephensi *in guinea-pigs.*

Average Weight in Grammes.	Number of Animals.	Dose in Milligrammes per 100 Grammes.	Results.
194	1	0·1	Died in 3½ hours.
238	10	0·08	One died in 1 hour 42 minutes, one in 1 hour 55 minutes, one in 2 hours 52 minutes, one in 4¼ hours, one in 5½ hours, one in 7 hours, one in 8½ hours, one in 11½ hours, and two in less than 21 hours.
226	4	0·06	One died in 13 hours, and three survived.
233	4	0·04	All survived without symptoms.

The lethal dose was 0·08 milligramme per 100 grammes, or 0·8 milligramme per kilogram, the

venom having rather less than half the toxicity of that of *Hoplocephalus bungaroides* for guinea-pigs.

The symptoms were predominantly respiratory. There was dyspnœa and the nose and mouth were wet. The animals raised their heads in the effort to get air into the thorax. Later there was collapse, weakness or paralysis of the limbs and definite "starting movements", which were probably asphyxial.

At autopsy there was invariably extensive hæmorrhage round the site of the injection. The lungs were blown up and overlapped the heart and were frequently the seat of extensive hæmorrhage. The blood was fluid and the coagulation time was greatly delayed. The intestines and adrenals were congested. In the female the uterus and adnexa were congested. There was no evidence of hæmolysis, and hæmoglobinuria was not observed.

The Hæmolytic Power of the Venom of Hoplocephalus Stephensi.

Miss Williams tested for me the hæmolytic power of the venom of *Hoplocephalus stephensi*, using 3% washed corpuscles of horse, rabbit and guinea-pig and defibrinated blood diluted to a corresponding extent. The volume of the system was 0·3 cubic centimetre: 0·1 cubic centimetre of corpuscles and 0·2 cubic centimetre of saline solution containing venom from 0·1 milligramme to $0{\cdot}1 \times 10^{-6}$ milligramme.

With washed horse red blood cells there was no hæmolysis; with horse defibrinated blood there was a trace of hæmolysis with 0·1 milligramme to 0·00001 milligramme of venom after twenty hours. With rabbit washed cells and defibrinated blood there was strong agglutination after twenty hours with a concentration of venom of 0·1 milligramme, but no hæmolysis. With guinea-pig washed cells 0·1 milligramme caused complete hæmolysis in four hours, 0·01 complete hæmolysis in twenty hours, 0·001 milligramme incomplete hæmolysis in twenty hours. With defibrinated guinea-pig blood 0·1 gave a trace of hæmolysis only in twenty hours.

The hæmolytic power of the venom is very feeble. It is in this respect only a little more active than that of *Hoplocephalus bungaroides*. No hæmolysis or hæmoglobinuria was observed in the rabbits and guinea-pigs which received the venom subcutaneously.

The venom resembles that of *Hoplocephalus bungaroides* in its local action at the site of injection, in the feebleness of its hæmolytic power, in the possession of a powerful thrombin, and in its toxicity as measured by its action on the peripheral nervous system, causing death by respiratory failure.

The relatively small size of the venom yield given by a full-sized snake makes it unlikely that the bite would cause a fatal result in man or a large animal unless a vessel were by chance entered by the fangs.

Acknowledgements.

My thanks are due to my colleague Miss F. E. Williams for the observations on the hæmolytic power of these two venoms, and to my one-time colleague Mr. Donald Thomson for the description and photograph of *Hoplocephalus stephensi*.

References.

(1) J. R. Kinghorn: "The Snakes of Australia", 1929, pages 191-193.
(2) C. H. Kellaway: "Observations on the Certainly Lethal Dose of the Venom of the Common Brown Snake (Demansia Textilis) for the Common Laboratory Animals", THE MEDICAL JOURNAL OF AUSTRALIA, December 12, 1931, page 747.

Reports of Cases.

A CASE OF OVARIAN HÆMORRHAGE.

By R. ERROL MAFFEY, M.B., B.S. (Sydney),
Singleton, New South Wales.

ON March 19, 1934, a married woman, aged twenty-nine years, consulted me. She stated that she had had a normal menstrual period commencing about December 30, 1933, and lasting four or five days. Menstruation occurred next on February 1, was more profuse than usual, and persisted for seven days, when there was an interval of two or three days without hæmorrhage, followed by a reappearance of the flow, but much less in amount; this had continued without remission to the time of consultation. About the time at which the vaginal hæmorrhage reappeared, the patient began to experience pain of a constant nagging character in the lower part of the abdomen, chiefly on the left side.

She had one child, aged seven years, and had had no miscarriages. Appendicectomy and "the womb stitched in place" (probably internal shortening of the round ligaments) had been performed eight or nine years previously in another town.

When entering the room, the patient walked in a stooped, restrained attitude, with the left hand holding the left side of the abdomen. The temperature was 36·7° C. (98° F.), pulse rate 86, and respiration rate 22 per minute. There was generalized tenderness of the lower part of the abdomen, most acute over the left iliac fossa, but no hyperæsthesia or muscular rigidity. On vaginal examination the cervix pointed downwards and backwards, was hard and irregularly lacerated, and the external os barely admitted the tip of the finger; the body of the uterus was difficult to define, but was not enlarged; no marked tenderness was elicited on tipping the cervix; pain was complained of during examination of all fornices, and there appeared to be a small, tender mass in the left broad ligament. A small amount of semi-fluid blood was present in the vagina.

A provisional diagnosis of "leaking" tubal pregnancy was made and the patient was sent into hospital for observation. During the next three days there was a continuous small loss of blood *per vaginam* and there was no change in the physical findings. On March 21 the temperature rose to 37·4° C. (99·4° F.) and the pulse rate to 120, and an increase in the low abdominal pain was complained of, though no marked difference in the patient's general condition could be observed. On March 23 vaginal examination was made under open ether anæsthesia administered by Dr. H. L. Stafford. The findings were identical with those at previous examinations, except that the right ovary and tube were thought to be enlarged and thickening of the left broad ligament was evident. Curettage of the uterus was then performed; but no placental tissue or any abnormality of the uterine wall (such as a submucous fibroid) was detected. Posterior colpotomy was debated, but it was decided to proceed immediately with exploration of the abdomen, which was opened through the scar of the previous operation, in the

mid-line below the umbilicus. A small amount of dark fluid blood was encountered on incising the peritoneum. The left Fallopian tube and ovary and the uterus were normal. The right ovary was larger than the left, measuring about five centimetres (two inches) from pole to pole, and 2·5 centimetres (one inch) in width and thickness. Adhering to its outer pole was a mass of friable blood clot, almost the same size as the ovary, partially covered by a thin membrane and attached also to the fimbriæ of the right tube. The condition was thought to be one of ovarian pregnancy, so the ovary and attached blood clot were removed, the normal right tube being left *in situ*. Some small masses of more recent blood clot were swabbed from the pouch of Douglas and the abdomen closed without drainage.

Convalescence was uneventful and the patient has menstruated normally and regularly since her discharge from hospital on April 10.

The specimen was examined by Dr. A. E. Finckh, whose report read as follows:

> The blood clot shows some small-celled infiltration (organized blood clot, appearing as if it had been there for some time). The ovary shows a large number of cysts, some very small, others distended with thick material—an adenomatous condition. There is no evidence of pregnancy or malignancy.

In a further communication Dr. Finckh stated that he could not indicate the origin of the hæmorrhage.

Comment.

Reports of actual cases of ovarian hæmorrhage are very difficult to find, and I wish to thank Miss Whitfeld, librarian of the New South Wales Branch of the British Medical Association, for procuring for me the only two reports which I have been able to consult. In both these cases the onset was acute and sufficient intraperitoneal hæmorrhage had occurred to cause symptoms of shock. In neither was hæmorrhage *per vaginam* noted. In one[(1)] a bleeding point was observed in what was thought to be a *corpus luteum* cyst. In the other[(2)] the pathologist reported "hæmorrhagic *corpus luteum* and follicular cysts; interstitial hæmorrhage and chronic oophoritis". Microscopic examination of the ovary from my patient revealed only a purely adenomatous condition, so that one must conclude that hæmorrhage occurred into one of the small cysts, which ruptured, allowing blood to escape into the abdominal cavity.

Acknowledgement.

My thanks are due to Dr. J. E. Wiseman for his helpful cooperation in trying to arrive at a diagnosis and for his assistance at the operation.

References.

(1) A. Verbrugghen: "Hæmorrhage from a Corpus Luteum", THE MEDICAL JOURNAL OF AUSTRALIA, August 27, 1927, page 296.
(2) Honoria Acosta-Sison: "A Case of Ovarian Hæmorrhage Simulating Ectopic Pregnancy", *Journal of the Philippine Islands Medical Association*, June, 1930, page 248.

Reviews.

PROGRESS IN PSYCHIATRY.

IN the second edition of "Recent Advances in Psychiatry" Henry Devine has revised and enlarged his earlier work and makes a critical review of psychiatry at the present day.[1] He does not attempt to define clinical entities, but deals with the subject in a wide manner and discusses fundamental principles. In an introductory chapter he stresses the point that general medicine is concerned with somatic reactions while psychiatry has to do with psychosomatic reactions.

Part I is devoted to the discussion of predisposition to mental disease, the author concluding that hereditary tainting and germinal injury are fundamental factors in the causation of mental disorder.

In Part II Dr. Devine considers in detail the various toxic infective and somatic factors in the causation of mental disorders. Febrile infection may be accompanied by symptoms of the nature of delirium, hallucinatory states or confusion which are benign, but they may also release latent schizophrenic or cyclophrenic psychoses. A valuable summary is given of the methods of treatment of confusional states. Attention is drawn to the fact that profound emotion or protracted anxiety may produce confusional states indistinguishable from those produced by organic factors. After reviewing the work and claims of Cotton and the toxic school, the author states that their claims are exaggerated, but that of causal factors in psychoses none is more worthy of research than chronic sepsis. The treatment of general paralysis of the insane by malaria is fully considered. On the basis of case reports it would appear that with this treatment 30% of patients are able to return to ordinary life, 30% die, and 40% remain chronic dements in hospitals.

The author describes the grave character deviations met with in children as sequelæ of encephalitis. Some writers see a resemblance between these states and schizophrenia, but in the latter the rigidity is psychogenic. In this connexion reference is made to the action of bulbocapnine in producing catatonic-like states of rigidity in laboratory animals.

Part III is devoted to consideration of reactions of psychotic subjects to somatic diseases. Many observers have reported resolution even of grave psychoses following intercurrent illness and on the basis of these experiences has been introduced shock therapy, which the author discusses in its various modes of application as well as allied methods of treatment, such as cerebral stimulation by concentrated carbon dioxide, prolonged narcosis, and etherization.

The physiological reactions in psychoses are discussed in four chapters under the headings of blood sugar, basal metabolism, deficient oxidation, and psychogalvanic reactions.

The biogenetic psychoses—manic-depressive psychoses, and schizophrenia—are described in detail and the author proceeds to discuss Kretschmer's work on physique and character.

Psychogenesis is considered in Part VI and the author gives an interesting summary of Freud's and Jung's psychopathological theories. In conclusion he gives a short review of psychotherapy as applied to psychoses. This book provides a critical review of psychiatry. Its language is plain and clear and both the specialist and the general practitioner will find it a valuable source of information. To each chapter is appended a bibliography. The one blemish of the book is the large number of misprints.

DERMATOLOGY.

R. CRANSTON LOW states that his book, "The Common Diseases of the Skin", is based on the lectures which he has been giving to his students.[1] This is the second edition.

In the introductory chapter there is embodied a description of the anatomy, physiology, pathology, and bacteriology of the skin, together with the general principles of treatment.

The remainder of the book is devoted to a description of the common diseases of the skin. It is to be regretted that no section has been allotted to syphilis, without which no book on diseases of the skin can be complete.

The number of illustrations has been more than doubled in this edition, and it now contains 150 plates, of which eight are in colour.

The publication should be of value to students with its short and lucid descriptions. It is well printed.

[1] "Recent Advances in Psychiatry", by H. Devine, M.D., B.S., F.R.C.P.; 1933. London: J. and A. Churchill; Australia: Angus and Robertson. Demy 8vo., pp. 375. Price: 19s. net.

[1] "The Common Diseases of the Skin: A Handbook for Students and Medical Practitioners", by R. C. Low, M.D., F.R.C.P.; 1934. Edinburgh: Oliver and Boyd. Crown 8vo., pp. 331, with illustrations. Price: 12s. 6d. net.

The Medical Journal of Australia

SATURDAY, AUGUST 25, 1934.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction, are invited to seek the advice of the Editor.

THE FEDERAL COUNCIL AND FUTURE CONGRESSES.

The meeting of the Federal Council of the British Medical Association in Australia is always a noteworthy occasion. At the meeting to be held in Sydney before the end of this month many momentous questions will be discussed; none will be more important than the discussion on the regulations governing the holding of congresses. At the last meeting of the Federal Council a subcommittee was appointed to consider the revision of the regulations and to bring forward suggestions to the next meeting.

In the ordinary course of events a congress is held in one place at intervals of about thirty years. There are six capital cities in the Commonwealth of Australia and four cities in the Dominion of New Zealand, in all of which are members of the Association who would be capable of running a congress and who would no doubt be anxious to have an opportunity of serving the profession. The result of migration of the congresses is that the organizers of each session find themselves without experience and are sometimes at a loss to know where to make a beginning with their plans. Of course ready help is always given by those who have held office at the previous session, but this help does not necessarily provide for uniformity in method. Even if necessary assistance regarding arrangements and so on is forthcoming, the president is entirely new to his duties and no doubt feels that he should have at his elbow someone experienced in congress methods, who could direct his attention to mistakes made by previous presidents, and who could advise him how those mistakes might be avoided. At the present time the only person who has an *ex officio* seat on the executive committee of all congresses is the Editor of The Medical Journal of Australia. Any help that he can give is connected more with the choice of papers, with the method of handling them and with the publication of the "transactions" than with the details of routine management of a congress. In our opinion, the Secretary of the Federal Council should be an *ex officio* member of the executive committee of all congresses. This statement needs careful explanation. Not for one moment do we suggest that secretaries of congress have shown any lack of efficiency or that the executive committee has ever failed in carrying out its duties, but we do think that they have often been put to trouble and subjected to worries that might, and should, have been spared them. The Secretary of the Federal Council in the circumstances suggested would have in effect an advisory position. He would not have to bear the brunt of the organizing work; this will always have to be done by the secretaries of the congress. He would be a kind of advisory master of ceremonies. Particularly would he have to give moral support to the president in his efforts to enforce the rules of debate in plenary sessions. A president, new, as he always is, to his duties, is diffident in restricting speakers at plenary sessions to the prescribed twenty minutes; if he had the constant reminder of the Secretary of the Federal Council of the uselessness of excluding discussion by allowing over-ardent speakers to exceed their time limit, he would overcome his diffidence. By his advice the Secretary of the Federal Council would secure uniformity in the management of successive congresses.

There are one or two other aspects to which the Federal Council should turn its attention. At the

conclusion of each session of congress there should be a plenary session at which the resolutions adopted by the several sections for transmission to the Federal Council might receive the *imprimatur* of the whole body of members. At present individual sections send recommendations to the Federal Council; this is wrong. The Federal Council should be approachable only by the whole congress and not by any section of it. Again, we hope that at this meeting the Federal Council will consider the formation of a section of comparative medicine. This has been repeatedly advocated in these columns and is long overdue.

The suggestions made herein for the consideration of the Federal Council show merely one or two directions in which improvement can be made. The Council may or may not see fit to adopt them; in any case its members know that they have the confidence of the members of the Branches; they also know that they must build for the distant future as well as for the needs of the immediate present.

Current Comment.

THE MORTALITY FROM HEART DISEASE.

PROBABLY all subjects of public interest are explored by statisticians, and in some cases, happily not those related to the medical sciences, the propagandist zeal of the inquirer overrides his accuracy. But in all statistical research it is necessary to be equipped with more than diligence and sincerity of purpose; the statistician is, or should be, more or less of a specialist, for he requires a combination of mathematical ability and technical knowledge of the subject he investigates. In medical inquiries one well recognized difficulty is the institution of "fashions" in diagnosis. The word is perhaps unfair, for these "fashions" are often due to real advances in knowledge, but fallacies may arise in two ways. Over-enthusiasm may cast too many cases into the new category; also even the fact of an increase in diagnostic accuracy makes the utilization of the previous figures very difficult. Alfred E. Cohen and Claire Lingg, in reviewing the subject of heart disease from the point of view of public health, remark on this difficulty.[1] They point out, for example, that the publication of papers on coronary occlusion by J. B. Herrick and others caused a sharp increase in the number of cases recognized in the Presbyterian Hospital in New York. This experience has been paralleled all over the world. Another certified cause of death that has suffered great alteration in numbers is senility, and, as recently pointed out in this journal by J. V. Duhig, as pathological knowledge grows, this inexact category rightly becomes less popular in mortality figures.

The purpose of the inquiry made by these authors is to add to the information available concerning the problem of cardiac disease as a mortality factor in a community. Since 1926 the subject has been studied from this angle, and the present report is particularly concerned with the apparent increase in deaths from cardiac disease in recent years. One of the authors, in a report made in 1926, pointed out that the curves relating to infectious and heart diseases had moved in opposite directions, the former falling, the latter rising. A considerable period of time separated these graphic changes; seventeen years elapsed between the fall in the infections mortality rate and the rise in the cardiac rate. This was interpreted as representing the natural course of events, for persons surviving the risks of infections early in life, though they may fall victim to cardiac disease later, will do so only at the appropriate age period. It was also observed that the increased rate of death from heart diseases involved persons over the age of forty; this is as expected, for the rate increases with each decade.

Now that the figures used in this study have been supplemented by those of more recent years, it is found that the deaths from heart disease of persons under forty still tend to fall, whereas those after forty tend to rise. The infectious diseases are responsible for fewer deaths in successive years, and this fall affects every decade. Now it is just in these figures that a fallacy lurks. The authors point out that it would be easy to assume that freedom from infectious disease resulted in a tendency for more deaths from cardiac and other causes to occur later in life. It has also been assumed by some writers that though less people die from diphtheria, scarlet fever and other acute infections in early life, the survivors may sustain some cardiac injury that will predispose to death in the later decades. There are, however, no adequate morbidity statistics available, and proof or disproof of this hypothesis cannot be attempted at present. Reverting to the survivors of the perils of infection, it is found that up to the sixth decade the saving from infectious disease is more than enough to account for the increased mortality from heart disease, but in the later years of life this is not so. Here comes in the question of more accurate diagnosis, whether allied to "fashions" or not. There seems to be warrant for the statement that when this greater exactness of certification is taken into consideration, there has been no really sharp increase in the cardiac deaths.

To sum up, it is found that when the cardio-renal disease groups are studied as a whole, the increases in death rate due to these causes in the later decades of life are definite but not alarming. There has been a definite decrease in mortality rates from infectious disease, and this affects not only the earlier decades,

[1] *The American Heart Journal*, February, 1934.

but the later also. This fall has largely helped to lower the death rate in all decades. Therefore there is no warrant to assume that there has been a consistent rise in the deaths due to heart disease as a result of the added strain of life, for other causes quite satisfactorily account for the somewhat misleading rise in the graphic curves. All this may seem rather dry, but in reality it is full of interest. It is useful to know that infections are proving less lethal throughout the span of life, and if escape from one peril confronts us with another, our correct attitude is to regard this as a challenge.

VITAMIN *C* IN THE HUMAN PITUITARY.

The ductless glands continue to receive close attention at the hands of the analytical chemist. In many centres persistent efforts are being made to isolate chemically, and perhaps to synthesize, the hormones of the adrenal cortex, pituitary and sex glands, as has already been done with the adrenal medulla and thyreoid. The histochemical test of Szent-Györgyi has already proved the presence of a high concentration of vitamin *C* in the cortex of the adrenal. The test depends on the highly reducing powers of ascorbic acid when brought into contact for fifteen minutes with a 0·4% solution of silver nitrate. Gough and Silva found that the anterior lobe of the pituitary gland of man, the ox and the dog contained an even higher proportion of ascorbic acid than the adrenal cortex. The dog's *corpus luteum* was also found to contain the acid to an appreciable degree. Biological assay has confirmed these findings. Indeed, under trial, the ox pituitary is as rich in vitamin *C* as any of the natural botanical sources of the substance, such as mango. Applying the silver nitrate test to material from forty-two hospital autopsies, J. Gough and S. S. Silva found that the pituitary in man invariably stained darker than the adrenal, but that the intensity of the reaction varied in different individuals. This series has now been extended by Gough to one hundred examinations,[1] and the conclusion has been reached that the quantity present varies approximately with the degree of nutrition of the subject, being least in states of emaciation. A qualitative classification of the intensity of the reaction in relation to the diseases fatal to the subjects examined revealed some interesting facts. A strong reaction appeared in 38 of the 100 patients, in 34 of whom general nourishment of the body appeared normal. The majority had died of rapidly fatal illnesses, such as meningitis, pneumonia and septicæmia, lasting under seven days. A moderate reaction was encountered in 25 patients, in 16 of whom bodily nutrition was normal. Moderate wasting was observed in eight and severe emaciation (*tabes dorsalis*) in one. A slight reaction was described in 26 instances. Nearly all of these were instances of long standing disease, such as cerebral softening, cancer, Hodgkin's disease, gastric ulcer, abscess of lung *et cetera*, and in nearly all undoubted emaciation was present. Eleven of the 100 patients failed to give any reaction at all. Of these, eight were severely emaciated and two moderately so. In all the latter the illness was of over three months' duration. Gough made an attempt to determine the character of the reaction in the normal man by testing the pituitary in seven examples of rapidly fatal injury. In five there was a strong and in two a moderate reaction. Both the latter persons, however, were over sixty years of age. Gough concludes that the younger the individual, the stronger the reaction, though in the newly born only a moderate intensity was evident. There was no seasonal variation in the degree of the reaction corresponding to the availability of vitamin *C* in the general diet. In the performance of the test, however, the temperature must be standardized. These tests were all done at room temperature. The time interval between death, autopsy and the performance of the test was without significance, though it should be remarked that all bodies were kept in a modern refrigerator.

It has been possible to demonstrate the presence of vitamin *C* in the adrenal medulla of the ox, but only after previous treatment with lead acetate. The "negative" reactions, when given by the human pituitary, are not altered by such treatment, which is known as the Huszác modification of the Szent-Györgyi reaction. Lacking any confirmation by biological criteria, Gough will not deny that some vitamin *C* may be present in such glands. It does seem possible, nevertheless, to connect the weak reactions with a deficient food intake, whether or not emaciation itself has any direct influence.

Other work has already shown that the vitamin *A* content of organs generally is probably subject to the same factors as those governing the vitamin *C* content. A deficient reaction is probably to be detected in the pituitary long before any clinical sign of scurvy appears. If further investigation shows that more accessible tissues, such as the blood or the excreta, give reliable reactions of the Szent-Györgyi order, an early lack of vitamin *C* may become evident, and this would have a practical bearing in the treatment and understanding of the vague conditions of minor vitamin lack.

[1] *The Lancet*, June 16, 1934.

AN APPEAL.

The Honorary Secretary of the Medical Benevolent Association of New South Wales has forwarded a report of the activities of the Association for 1933. He points out that a sum of £425 was distributed in the year to beneficiaries and that £80 was disbursed as Christmas gifts. The association does not aim at building up large reserves, and depends on subscriptions and donations. Life membership is obtainable for £10 10s., and the annual subscription for membership is one guinea. The trustees of the fund appeal to members of the medical profession in New South Wales for continued support.

Abstracts from Current Medical Literature.

RADIOLOGY.

Bone and Joint Changes in Hæmophilia.

LEON SOLIS-COHEN AND SAMUEL LEVINE (*American Journal of Roentgenology*, April, 1934) report several cases of hæmophilia with hæmarthrosis and discuss the condition. The pathological changes occurring in the joint may be divided into three successive phases: (i) hæmarthrosis, (ii) panarthritis, (iii) the regressive stage. Hæmarthrosis is associated with thickening of the fibrous joint capsule, but is unaccompanied by bone changes. During this acute stage resulting from repeated hæmorrhage, the affected joints become swollen, painful and tender. The overlying skin presents a bluish discoloration resembling hæmatomata, and the surface temperature is increased. Since no clotting occurs, the pressure in the joint is tremendous, being equivalent to the systolic blood pressure of the general circulation. The bleeding distends and is limited by the joint capsule, and the blood, acting as an irritant, produces thickening and a chocolate discoloration of the synovial membrane. In the panarthritic stage definite vacuolization in the epiphysis above the articulating surface occurs, the edges of the latter being sharply etched and well defined. The opposing joint surfaces are separated by a hæmorrhagic effusion. These areas of decalcification may be due to pressure necrosis or to unknown hæmolytic agents acting as ferments and producing areas of bone dissolution. In the regressive phase the loss by destruction or absorption of cartilage is due to interference with the circulation and results in deformity. Pseudolipping is present, suggesting a true osteoarthritis. Films reveal the following diagnostic points: (i) Acute stage of hæmarthrosis. The effusion of blood casts a denser shadow than an effusion in an ordinary synovitis. The capsule is distended and the joint space widened. Bony changes are absent. (ii) In the panarthritic stage there is increased density or thickening of the joint capsule. There is often observed a crater-like excavation in the extraarticular portion of the diaphysis. Shadows of an organized blood clot are found in the recesses of the joint and follow the outline of the joint capsule. The density of synovial tissue may resemble that of calcification observed in *myositis ossificans*. Widened joint space occurs without bony ankylosis. In the knee the patella may float and the patellar ligament be bowed forward by a hypertrophied pad beneath it. The femoral condyles may be broadened. (iii) In the regressive stage the articular surfaces are irregular and the ends of the bone entering the joint may be deformed. Pseudo-osteophytes are observed, due to the hollowing out of the extraarticular surfaces of the articulating bones. True osteophytes are unusual. The spine of the tibia is often deformed and the intracondylar notch widened. In the elbow a subperiosteal hæmorrhage may undergo calcification. New bone formation may occur. In the hip bone changes may closely simulate those of Perthes's disease. In the shoulder the bone changes may simulate *caries sicca*. In the chronic stages this condition must be differentiated from hypertrophic osteoarthritis, tuberculosis, a Neisserian joint, and remotely a Charcot joint; in the acute phase, from acute articular rheumatism and any acute pyogenic infection or synovitis.

Excretory Urography.

W. F. BRAASH (*The Journal of the American Medical Association*, December 9, 1933) states that excretory urography will be of greatest practical value to clinical diagnosis in: (i) determining the presence of stasis in the renal pelvis or ureter, (ii) aiding in interpretation of shadows in the upper part of the urinary tract, and (iii) giving a fairly accurate estimate of renal function. Complete failure of visualization on one side in repeated films must be interpreted as indicative of renal dysfunction. Fairly clear visualization on one side and poor visualization on the other does not necessarily indicate a lesion. A difference in the degree of visualization of the two kidneys is often observed when no other evidence of change is apparent. There may be also a difference in the time of visualization, one pelvis being much better visualized in the first film and the other in a later film. There is also variation in the time of emptying on the two sides. Exact interpretation of moderate delay in visualization may be difficult. Renal ptosis usually can be determined without the aid of urography, but not the indications for operation. It is now generally recognized that unless there is definite evidence of renal stasis, nephropexy is usually not indicated. Excretory urography offers an ideal method for determining minor degrees of stasis. As a rule renal tuberculosis will cause some degree of deformity in the urographic appearance. Although the abnormality may not be typical of renal tuberculosis, nevertheless, with the aid of other evidence, such as the presence of acid-fast bacilli in the urine, a diagnosis frequently can be established. When any of the following signs are observed in the urogram the diagnosis of renal tuberculosis often can be inferred: absence of or marked delay in visualization, irregular pyelectasis or ureterectasis, failure of visualization of one or more calyces with irregularity of pelves, or definite necrosis on one side and a normal pelvis and ureter on the other. It should be stated that normal visualization on one or both sides would not exclude renal tuberculosis. Excretory urography should be employed as a routine when injury to the urinary tract is suspected. Although reflex inhibition of excretion following severe injury may interfere with interpretation, the character and extent of rupture of the kidney may often be visible in the urogram. Rupture of the bladder may also be determined by the method better than by any other and without the dangers of secondary infection. Excretory urography may also be of value in making an approximate estimate of the amount of residual urine in the bladder, when it is impossible or inadvisable to employ a urethral catheter.

X Rays as an Aid in the Diagnosis of Hæmophilia.

EDWIN L. REPPINS (*American Journal of Roentgenology*, May, 1934) reports characteristic changes in the Röntgenograms of the knees in three cases of hæmophilia. The most striking change is widening and deepening of the intercondylar fossa. Other changes are narrowing of the joint space and marked osteoporosis of the bones adjacent to the joint. There are generally some arthritic changes present. The explanation of the widening of the intercondylar fossa is uncertain. It may be that the bleeding occurs at the point of attachments of the crucial ligaments at the apex of the fossa. Bleeding causes bone deterioration, and it is conceivable that the pressure from the bleeding would cause erosion at this point, since at the apex of the intercondylar fossa there is no cartilage. However, it is possible that intraosseous hæmorrhage plays an important part.

Thoracic Stomach.

H. W. GOODALL AND L. H. HOYT (*Archives of Internal Medicine*, April, 1934) discuss the condition of thoracic stomach and report five cases. The condition is not uncommon and is due to a congenital foreshortening of the œsophagus which prevents the descent of the stomach from the thorax, where it normally develops into the abdominal cavity. The condition has been usually overlooked, since the presence of the stomach or a part of the stomach in the thoracic cavity has been considered to be a hernia of the stomach through the œsophageal opening of the diaphragm. In these cases the part of the stomach lying above the diaphragm does not become reduced in the erect position or after emptying of the stomach, since it is held up by the foreshortened œsophagus.

X Ray Examination of Laryngeal and Hypopharyngeal Tumours.

BORGE WORNING (*Acta Radiologica*, February, 1934) states that tumours in the larynx and hypopharynx manifest themselves radiographically chiefly in three ways, namely: (i) by projecting into the lumen of those organs, (ii) by dislocating the larynx, (iii) by destroying the laryngeal cartilages. If a tumour projects into the lumen of the larynx or hypo-

pharynx, it will be visualized as a sharply contoured soft tissue shadow. On the other hand, if the tumour is situated in the surrounding soft tissues, it will, when sufficiently large and circumscribed, stand out as a definable soft tissue shadow. If a tumour lies outside the larynx and is of sufficient size, it will displace it as a rule anteriorly. If a tumour extends its growth into an ossified laryngeal cartilage, the result will be on the one hand disintegration, on the other hand neoformation of bony tissue. The X ray appearance will present a mixture of destructive processes expressing themselves in numerous irregular rarefactions with ragged, uneven contours, and neoplastic processes manifesting themselves by neoformation of bone and small calcifications. If the destruction is only slight, diagnosis will be difficult, especially if the physiological ossification is not complete.

PHYSICAL THERAPY.

Actinomycosis.

R. STEWART-HARRISON (*The British Journal of Radiology*, February, 1934) gives a schematic classification of various methods of X ray treatment worked out by Schinz, as follows: (i) disjuncted fractional treatment, (ii) simple fractional treatment, (iii) protracted fractional treatment, (iv) saturation treatment, (v) method of decreasing doses. Treatment was carried out in thirty cases verified microscopically. Twenty-two of the patients had the disease localized to head and neck; all were quite cured. The results by the disjuncted fractional method and protracted fractional method are contrasted; the latter is found to be the better. The author suggests that surgical methods should be limited to aspiration, and small incisions of abscesses and laparotomy with biopsy.

Carcinoma of the Œsophagus Treated by Radiation.

WALTER M. LEVITT (*The Proceedings of the Royal Society of Medicine*, February, 1934) acknowledges that the results previously obtained in treatment of carcinoma of the œsophagus by X rays have been bad, although in rare instances great improvement has been recorded. The fact that the histological characters of these growths put them in the radio-sensitive class makes the disappointment greater. The difficulty may be that sufficient dose is not delivered at depth, so Dr. Levitt has attempted to rectify this by using multiple long narrow fields placed at intervals round the chest wall. Daily doses are given over five to six weeks. He has treated eight unselected cases by this method. They have all been treated during 1933 and therefore, although seven patients are alive and well, the period is short; nevertheless the results are very encouraging, because subsequent œsophagoscopies have shown complete absence of growth. The author gives the case notes and states that the so-called fibrous stricture following X ray treatment of carcinoma of the œsophagus should always be regarded with the gravest suspicion, as contracted scars are not seen after X ray treatment alone in other situations.

Malignant Neoplasms of the Thyreoid Gland.

B. F. SCHREINER AND W. T. MURPHY (*Annals of Surgery*, January, 1934) show that malignant disease of the thyreoid gland is increasing and now represents 0·37% of all malignant disease seen. Both carcinoma and sarcoma are included in this series. Forty-two cases are included in this article, all but one of which were treated by means of irradiation either after or without surgery. Twelve patients were treated with X rays alone, of whom two are alive between one and two years later. One patient with lymphosarcoma, from whom a biopsy was made and who was treated by X rays alone, is alive fifteen years later. In the remaining cases the disease was so far advanced that no biopsies were made; irradiation was given apparently as a palliative only. The authors conclude with the statement that it would seem that the disease, with few exceptions, is a fatal one and that it would seem that the only curative procedure would be early operation followed by irradiation.

Reaction to Radiation in Lymph Glands Containing Carcinoma Metastases of the Squamous Type.

LEONE MCGREGOR (*Acta Radiologica*, April 15, 1934) describes the reaction of large doses of X rays and radium on non-cancerous and cancerous lymph tissue. The non-cancerous glands enlarged and showed hyperplasia with large, prominent, œdematous follicles containing many mitoses and sometimes miliary hæmorrhages. The carcinomatous glands showed massive central necrosis which was in direct proportion to the amount of radiation. The effect was undoubtedly of vascular origin and must be regarded as anæmic necrosis. A favourable radiation effect (marked regression of the tumour) was accompanied by a favourable reaction of the tissues adjacent to the tumour. This consisted of an active proliferation of the connective tissue, proliferation of young fibroblasts, exudation of lymphocytes, leucocytes and plasma cells, upgrowth of new capillaries, and formation of granulation tissue. The tumour mass was broken up by invading connective tissue, and the individual tumour cells underwent degenerative changes consisting of swelling of the cytoplasm, hydropic degeneration of the cell bodies, hornification, calcification, cell atrophy and complete necrosis. In several specimens no gross or microscopic evidence of tumour tissue could be demonstrated. As far as could be judged from a very great number of sections of each specimen, glands from eleven cases (seven lip, two tongue, one buccal cavity and one vulva) showed apparently complete destruction of the cancer cells. Nothing of the cancer remained except an empty cavity or a cavity containing necrotic cornified material and lined by giant cells of the foreign body type. Twenty-three glands contained large areas of cancer necrosis lined by cancer epithelium. In many cases a large or small part of the cyst wall was formed by giant cells. The lesions seen in the cancer tissue of the lymph glands in this group are exactly duplicated in a few of the non-radiated cancerous lymph glands. In these non-radiated glands, however, there were usually, but not always, more mitoses. Giant cells of the foreign body type were always found in radiated and non-radiated glands wherever necrotic cancer came directly in contact with lymph glandular tissue. Where the necrotic cancer was surrounded by living cancer cells, no giant cells formed. Two-thirds of the radiated cancerous lymph glands showed changes in the blood vessels. The most marked lesions were seen in the small and medium sized vessels of glands removed about two months or more after radiation. The blood vessel changes seem to be dependent on the presence of cancer in the gland since there were no vascular lesions in the radiated non-cancerous glands removed in the same group with cancerous glands which did show vascular lesions. There was no difference in the amount or degree of vascular change following radium and X ray treatment. In Case XXVIII, where the left inguinal glands were given Röntgen therapy and the right inguinal glands radium, the histological picture was exactly the same in all respects.

Diathermy in Ambulatory Gynæcological Patients.

L. E. FRANKENTHAL AND A. J. KOBAK (*Archives of Physical Therapy, X-Ray and Radium*, April, 1934) discuss the results of treating thirty infections of the cervix and parametrium by medical diathermy. Local medications and home irrigations were employed as usual, and diathermy treatments were an adjuvant to the usual routine. The results are classified as follows: Of patients with localized Neisserian infection three were cured, two were improved; of those with pelvic adhesions three were cured, eight were improved, one was not improved; of those with pelvic inflammations three were cured, eight improved and four not improved. Also the authors used surgical diathermy in thirty cases of erosion of the cervix. Healing after one to two years was found in twenty-seven of the thirty cases. The authors claim that this method compares favourably with treatment by thermo-cautery and that the need for plastic surgery was avoided in several instances.

Special Articles on Treatment.

(Contributed by request.)

XXXVIII.

INFECTIONS OF THE ANTERIOR ASPECT OF THE HAND.

PERHAPS nowhere in the whole realm of surgery is an exact knowledge of the detailed anatomy, surface and deep, more necessary and of more practical utility than in the treatment of infections of the hand.

This is sufficient excuse to digress briefly to recall the more important landmarks of the hand and their deep relations.

When one examines the palmar surface of a hand, one sees that there are innumerable lines on it; the coarser and more conspicuous are flexure or palmist's lines, the finer and less obvious are papillary ridges or finger print ridges. The flexure lines are surface evidence of the deeper-seated joints, in fact they are, as Wood Jones has so aptly said, the "skin joints", and at these points the skin of the fingers and hand is comparatively fixed by the blending of the skin, superficial and deep fascia, so that flexure lines very closely resemble scars resulting from clean incised wounds.

The more evident flexure lines are the distal, middle and basal creases of the fingers, the distal transverse, the proximal transverse, and the radial longitudinal lines of the palm, and at the base of the palm the bracelet creases of the wrist which, typically, are two in number.

Each of these bears a constant relation to the deeper structures, and these relations one must now briefly enumerate.

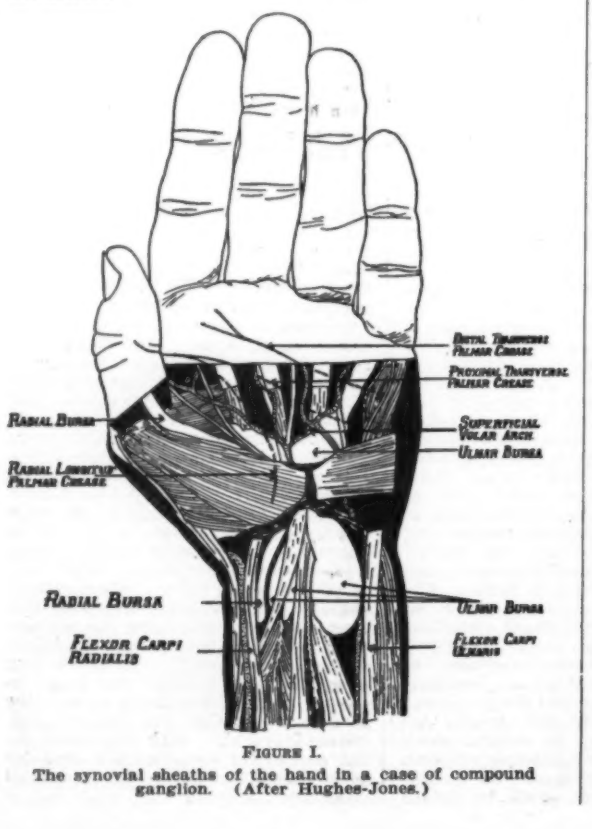

FIGURE I.

The synovial sheaths of the hand in a case of compound ganglion. (After Hughes-Jones.)

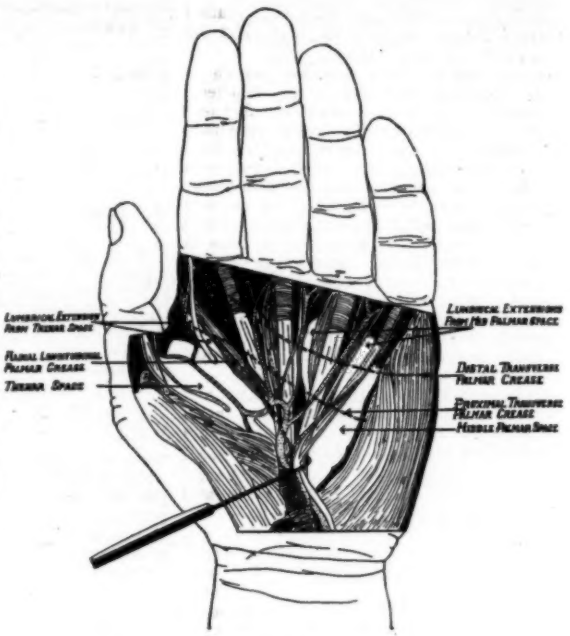
Lumbrical Extension from Thenar Space
Radial Longitudinal Palmar Crease
Thenar Space
Lumbrical Extension from 1st Palmar Space
Distal Transverse Palmar Crease
Proximal Transverse Palmar Crease
Middle Palmar Space

FIGURE II.

The thenar and the middle palmar spaces of the hand and their lumbrical extensions. (After Hughes-Jones.)

The distal digital crease is just proximal to the distal interphalangeal joint, the distal line of the middle digital crease almost exactly marks the line of the proximal interphalangeal joint, the basal digital crease lies at the same horizontal level as the web of the fingers and about 18 millimetres (three-quarters of an inch) distal to the metacarpal phalangeal joint; it also marks the junction of the upper and middle thirds of the proximal phalanx.

The distal transverse line of the palm on its ulnar side crosses the necks of the metacarpal bones and limits proximally the synovial sheaths of the fingers; it also lies opposite the division of the middle part of the palmar fascia into its digital processes (see Figures I and II).

The proximal transverse line of the palm at the level at which it crosses the metacarpal bone of the ring finger, limits distally the ulnar bursa and approximately marks the position of the superficial palmar arch.

A line joining the radial end of the proximal transverse crease with the ulnar end of the distal transverse crease marks the distal limit of the thenar and middle palmar spaces (see Figure II).

The radial longitudinal line varies in its position with the development of the thenar muscles; as a rule it passes to the ulnar margin of the metacarpal of the middle finger, and its proximal portion overlies with peculiar constancy the joint between the greater multangular and lesser multangular of the distal row and the navicular of the proximal row of carpal bones. Another prominent line of the palm is the medial longitudinal line, and this, together with the ulnar longitudinal line, bears testimony to the very great importance to function of the transverse arch of the hand.

The proximal transverse crease of the wrist corresponds accurately with surface projection of the joint between the lower end of the radius and the upper surfaces of the navicular and lunate bones of the proximal row of the carpus.

The distal transverse crease of the wrist marks exactly the joint between the lunate and the triquetum and *capitatum*. It also corresponds to the proximal margin of the transverse carpal ligament. Thus the lunate bone may be represented as lying in the middle line of the forearm between the two bracelet creases.

It may be added that the main crease lines are developed very early in fœtal life, but may be altered both by occupation and disease.

The superficial palmar arch, which is superficial to the flexor tendons and lumbrical muscles, lies in the same horizontal line as the distal border of the fully abducted thumb, which line falls a little proximally to the proximal transverse palmar crease, where the latter crosses the middle metacarpal bone. The deep volar arch, which lies deeply in the palm on the proximal ends of the metacarpal bones and on the origin of the interosseous muscles and deep to the long flexor tendons and their synovial sheaths, is from 12 to 18 millimetres (half to three-quarters of an inch) proximal to the superficial volar arch.

On inspection of the palm of the hand, besides the lines, are noticed several elevations and depressions of which the most prominent are the thenar and hypothenar eminences, caused by the small muscles of the thumb and little finger respectively, they form the lateral boundaries of the hollow of the hand which is limited distally by the depressions at the bases of the fingers caused by the fusion of the digital processes of the palmar fascia with the synovial sheaths containing the flexor tendons.

Between these depressions are three elevations opposite the webs of the fingers; they are formed by fat, in which are embedded the digital vessels and nerves and the lumbrical muscles.

The skin of the palmar aspect of the fingers and hand probably varies more in texture with the occupation of its owner than that of any other part of the body. It contains sweat glands, but, on account of the total absence of hair from this region, no sebaceous glands. The sweat glands open at the summit of the papillary ridges and, as Wood Jones has said, these ridges may be regarded as the nipple-like orifices of the ducts fused in linear series. On account of the use made of the hand, it is reasonable to presume that it is more liable to trauma and subsequent infection than any other part of the body.

Let us now enumerate the chief types of infection to which the volar aspect of the hand is prone:

1. Those involving the subcutaneous tissues, for example, whitlows and felons, subepithelial abscesses, collar button abscesses.

2. Infections of the tendon sheaths, of which, on account of their anatomical connexions, those affecting the tendon sheaths of the thumb and little finger are by far the most serious.

3. Lymphangitis.

4. Cellulitis of the hand, particularly when the middle palmar and thenar spaces are involved.

5. Infection of the major forearm space or Parona's space, which is most commonly secondary to tendon sheath infections of the thumb and little finger.

Boils and carbuncles of the volar surface of the hand are relatively uncommon owing to absence of hair follicles on the palmar aspect, but infections of the skin frequently occur from occupational injuries, particularly in the process of "hardening up" at the beginning of new employment, and these, if not cared for properly, may lead to infection of the cellular spaces.

Infections Involving the Subcutaneous Tissues.

Felons or whitlows are probably the most common infections of the distal phalanx; they generally follow a pin prick or some similar injury, noticed or unnoticed. The patient first becomes conscious of a stinging pain in the distal phalanx, which soon becomes throbbing in character and severe enough to prevent sleep. The distal portion of the finger becomes swollen and red and is very tender to the touch, particularly over the site of the origin of infection. The anatomy of this region explains the pathological findings. The distal four-fifths of the distal phalanx is really a closed space divided into numerous compartments by dense fibrous septa running between the skin and the periosteum of the distal phalanx. These compartments contain fat, sweat glands and blood vessels. The main blood vessels run parallel to the phalanx and, before entering the so-called closed space, give off a blood vessel supplying the epiphysis of the distal phalanx. The blood supply of the diaphysis of the distal phalanx comes from the rich arterial plexus of the pulp and enters the bone through relatively large nutrient foramina, so that it is easy for infection to spread into the diaphysis, either directly or indirectly, by infected arterial or lymphatic emboli from the pulp. It can be readily understood that any infection entering the closed or pulp space, either by way of the glands or from direct inoculation, will cause marked swelling and, on account of the denseness of the fibrous tissues, any increase in its content must exert pressure on the blood vessels and so tend to occlude them, thus leading to necrosis and death of the area they nourish. This arrangement of the blood vessels is the explanation of the frequency with which the greater part of the distal phalanx is sequestrated following felons improperly treated. The escape of the epiphysis or, in adults, of that part of the phalanx which corresponds to the site of the epiphysis, is due to its blood supply not being interfered with.

Early and adequate incision, not waiting until fluctuation is obtained, is the key to successful treatment. The incision, if the site of infection is known and if the infection is still localized, should be made over the site of the infection, particularly if it is still only subcutaneous. But if the whole pulp space is involved, the incision should be lateral—a straight incision, if necessary, on both sides of the distal phalanx. There is no need to prolong the incision distally—the hockey stick incision—as this extension results in a scar which interferes with the tactile acuity of the finger. This objection also holds against median incisions, which, moreover, are dangerous, because if prolonged too far proximally, they may lead to infection of the tendon sheath of the finger.

After incision hot foments are used or hot hypertonic saline baths, in which the whole hand is kept under hot water almost continuously until the dangers of extension are over.

Between bathings the finger should be immobilized on a splint and the hand carried in a scarf.

If treatment is delayed, and sometimes even when treatment is instituted early, infection spreads to the distal phalanx. If this occurs, it delays healing until the dead bone is sequestrated. Once the sequestrum is extruded or removed, the wound soon heals.

Subepithelial abscesses may occur on any part of the palmar surface of the hand. The diagnosis is generally obvious and the treatment is early removal of the entire elevated epithelial covering.

Collar button abscess (frog felon) occurs typically under the dermal and epidermal tissues of the distal edge of the palm. In those engaged in heavy labour the epithelium becomes markedly hypertrophied and is frequently injured by abrasions and cuts, the portals of entry for infective organisms. If the abrasions are only superficial, the lymphatics may carry the infection deeper; but if the cuts are deep, the inoculation may be direct into the deeper tissues. The sites of infection are most frequently opposite the webs of the fingers, for here the palmar aponeurosis is very thin and the underlying fat spaces may be involved along with the cellular tissue of the web. The infection may spread by continuity to the lumbrical canals and so infect the palmar spaces. These abscesses generally point on the dorsum between the bases of the fingers and demand early incision.

The patient should be given a general anæsthetic and a tourniquet should be applied to the arm. After the first loculus is opened careful search should be made for the opening of a second loculus. This can be done with a probe, but it is preferable to sponge the operative field free of pus and then to apply light pressure to the surrounding tissues, all the time watching carefully for the tell-tale bead of pus. If the tissues of the web are involved, the best incision is one extending from the palmar to the dorsal surface, slitting up the web freely. There may be abscesses in both the thenar and hypothenar areas beneath not only the skin, but also the deep fascia which covers these areas. These abscesses are as a rule small and are generally secondary to direct infection from puncture; and their treatment should occasion no worry, as the best results follow simple incision through the overlying tissues.

Infections of the Tendon Sheaths and of the Radial and Ulnar Bursæ.

The tendon sheaths are the tunnels of fibrous tissues lined with synovial membrane, in which the flexor tendons covered by synovial membrane are suspended by the "*vincula tendinum*", in which run the blood supply to the tendons. Since the tendon of the *flexor pollicis longus* and the tendons of the *flexor profundus digitorum* are inserted into the base of the distal phalanx of their respective digits, the tendon sheaths of all the digits end just distal to the distal crease of the finger. It is extremely important to keep constantly in mind how close the tendon sheath is to the skin at this point. At their proximal end, however, they end very differently. In the ring, middle and index fingers they terminate in loose areolar tissue in close proximity to the major fascial spaces of the hand. A line joining the radial extremity of the proximal transverse palmar crease with the ulnar extremity of the distal transverse crease accurately marks their extent proximally. This line is about a thumb's breadth proximal to the web of the fingers, and crosses the necks of the metacarpal bones.

In the thumb the sheath of the *flexor pollicis longus* communicates in the great majority of persons with the radial bursa, which lies superficial and radial to the thenar space and terminates proximally about 2·5 centimetres (one inch) above the proximal limit of the transverse carpal ligament, that is, 2·5 centimetres above the distal bracelet crease of the wrist.

The sheath of the flexor tendon of the little finger as a rule communicates with the ulnar bursa, which is a good sized sac covering over the ring metacarpal and extending radially as far as the middle metacarpal bone. It extends proximally under the transverse carpal ligament, comes to lie superficial to the *pronator quadratus*, and ends about 2·5 centimetres (one inch) above the distal bracelet crease of the wrist, namely, at the same level as the radial bursa. It does not completely surround the superficial and deep flexor tendons, but they are, as it were, invaginated into it from the radial side. In the palm the ulnar bursa lies superficial to the middle palmar space.

Tendon Sheath Infections.

The site of origin of infections of the tendon sheath is far more frequently on the volar surface of the finger than in the palm. The infection may be primary, following a prick of the volar surface of the finger, especially of the proximal interphalangeal crease. For at this site there is practically no subcutaneous tissue and, moreover, there may exist hernia-like protrusions of the synovial membrane through the theca. Or it may be secondary through lymphatic extension or direct continuity, for example, from an infective arthritis of the proximal interphalangeal joint or a periosteomyelitis of a middle phalanx, and at times from an ill-advised median incision of a distal closed space infection. On rare occasions the sheaths may be involved in the course of a systemic infection.

Signs and Symptoms.—General constitutional signs vary with the virulence of the infection and the constitutional resistance of the individual. The local signs are as follows: The whole finger is swollen symmetrically; this is the essential diagnostic point in differentiating tendon sheath infections from felons. The finger is held in the semi-flexed position; the patient will not actively extend the finger, though he may increase the degree of flexion. Passive extension is extremely painful, the pain being most marked at the proximal end of the sheath in the palm. Tenderness is most marked on pressure over the anatomical limits of the sheath, particularly on deep pressure at the proximal end of the sheath at the level of the metacarpal phalangeal joint. The latter is by far the most important sign and, if positive, is diagnostic. Absence of tenderness over the middle phalanx goes far to rule out tendon sheath infections. If rupture of the sheath has taken place, these signs and symptoms may be much less obvious. With infections of low virulence the inflammatory reaction may be limited by plastic adhesions to a portion of the synovial sheath, but this is a very rare occurrence. Complications of tendon sheath infections, seen late or inadequately treated, are infections of the related lumbrical spaces, and through these of adjacent tendon sheaths and fascial spaces, infective arthritis of the proximal interphalangeal joint, osteomyelitis of the middle phalanx, infections of subtendinous spaces.

In infections of the tendon sheaths of the little finger and thumb the ulnar and radial bursæ may be involved and through these Parona's space. In very late cases the wrist joint itself may be involved.

It can thus be seen that the anatomical relations make infection of the synovial sheaths of the little finger and thumb much more serious than similar infections affecting other fingers, on account of the risk of involvement of the ulnar and radial bursæ; for if either of these is involved and not promptly opened, the infection may quickly spread to the other, so leading to secondary infection of its associated tendon sheath.

Another complication of delayed treatment of infection of the radial bursa is infection of the wrist joint and osteomyelitis of the proximal end of the second and third metacarpal bones. It has been shown in many cases that this spread of infection is not direct, but through the synovial sheath of the *flexor carpi radialis*.

If there is delay in active treatment of infections of the ulnar and radial bursæ, they rupture into the major forearm space, known as Parona's space. This is the area of cellular tissue which lies between the *flexor profundus* in front and the *pronator quadratus* and interosseous membrane behind. Involvement of this area in the infection is characterized by increased swelling of the forearm and by brawny induration. If one is in doubt whether the space is involved, it should be drained.

Extension to the lumbrical spaces is by direct continuity and is characterized by redness and swelling of the tissue between the fingers in the dorsum of the corresponding web. Infection of these spaces may lead to secondary involvement of the middle palmar space, if infection first begins in the little, ring, or middle finger, and of the thenar space from infection beginning in the middle or index finger. Either of these fascial spaces may also be involved by direct rupture of their associated bursæ.

Treatment of Tendon Sheath Infections.—As in other infections of the hand, the factors which aid are hot fomentations or hot hypertonic saline baths, rest, plenty of fluids, light diet, and confinement to bed, good nursing and care. When tenderness is localized over the site of a tendon sheath, the sheath should be opened, and it is better to err on the side of haste rather than of delay. Operation should always be done under general anæsthesia; gas and oxygen is the ideal anæsthetic, if available. One has seen many tragedies, the result of use of local anæsthetics, in these infections.

A tourniquet should always be applied to secure a bloodless field, with its resulting free vision. If a blood pressure armlet is used it may be partially deflated and left on for some hours to produce a passive hyperæmia, with its resulting benefits, and to prevent a flooding of the blood stream with toxic material.

The incision should begin near the site considered to be the source of the infection; and the sheath should be opened at the side, not in the median line. Cut the length of the sheath on the proximal or middle phalanx, but do not incise the sheath opposite the articulation. If the skin incision is made too far in a lateral direction, the digital blood vessels may be cut; or if the incision should be posterior to the vessels, it is impossible to avoid dividing the branches running into the sheath. Incision in the skin should therefore be anterior to these digital vessels and nerves lying on either side—the nerves run slightly anterior and medial to the vessels. The first incision having been made, apply pressure over the rest of the anatomical extent of the sheath. As the operative field is bloodless, any escape of pus following this application of pressure is easily seen and so enables one to judge the necessity or otherwise of further incision.

As a rule, owing to the speed at which infection spreads within the sheath, a second similar incision over the proximal or middle phalanx should be made. If the infection is severe, make a longitudinal incision on each side of each phalanx. If the proximal part of the sheath is involved, a third incision is made, beginning over the middle of the sheath from the proximal digital crease,

extending a thumb's breadth into the palm of the hand. The skin incision should be carried slightly proximal to the end of the sheath so as to allow the sheath to be opened and freely drained at its bulbous proximal end. The skin incision may be continuous over the entire extent of the sheath, but the sheath should not be incised opposite the joints unless the condition is very grave, when a complete opening of the sheath is advisable (see Figure III).

FIGURE III.

From Kanavel's "Infections of the Hand", for comparison with Figure I. Lines show area of possible incisions for infection of the tendon sheaths. In case of doubt, the free incision of the whole sheath is to be advised.

In infections of the tendon sheaths of the thumb and little finger the question whether the infection has extended, involving the radial and ulnar bursæ arises. There is a small percentage of cases in which these tendon sheaths and bursæ do not communicate, or in which the opening is so narrow that it is closed by the inflammatory reaction. Generally, however, the possibility of them doing so makes the early diagnosis and treatment of these infections all the more urgent.

Infections of the Radial and Ulnar Bursæ.

Anatomy.—Deep to the transverse carpal ligament there are three synovial compartments. The medial compartment contains the four tendons of the *flexor sublimis digitorum* and the two differentiated portions of the *flexor profundus digitorum*. Above the upper border of the transverse carpal ligament the closed synovial sac extends as far up as the upper bracelet crease line on the volar surface of the forearm, whilst distal to the transverse carpal ligament this sheath spreads out in the palm of the hand as an extensive synovial sac, known as the ulnar bursa. The portion connected with the tendons of the index, middle and ring fingers extends distally as far as the middle of the palm at the line of the outstretched thumb. The portion in relation with the flexor tendon of the little finger is continuous as a rule with the tendon sheath of that tendon. The second compartment, which contains the *flexor pollicis longus*, extends proximally as far as 2·5 centimetres (one inch) proximal to the upper border of the transverse carpal ligament, and distally, as a rule, to the base of the distal phalanx of the thumb, but may end at the metacarpal-phalangeal joint.

The third compartment contains the *flexor carpi radialis* and is on a deeper plane than the other two compartments.

While under the transverse carpal ligament, the radial and ulnar bursæ are in very close proximity to one another, sometimes actually in open communication. Clinically they should always be considered as being in communication, as infection of one space quickly spreads to the other.

Infection of these bursæ is characterized by development of œdema of the hand, especially upon the dorsum; the palm looks full, but the concavity of the palm is maintained; this is in marked contrast to the loss of the palmar concavity and ballooning of the palm which occurs in fascial space infections.

The site of the greatest swelling is just proximal to the transverse carpal ligament, and the tightness of this ligament makes the swelling stand out in sharper relief. Extension to the ulnar bursa is diagnosed by the increased swelling of the hand, particularly on the dorsal surface, and by extension of the marked tenderness, characteristic of tendon sheath infections, to the site of the ulnar bursa. The point of greatest tenderness is just proximal to where the distal transverse crease of the palm crosses the hypothenar eminence. The thumb also becomes sensitive to movement, and this may be due to irritation of the radial bursa or to actual extension of the infection into the radial bursa. If the thumb becomes sensitive, it is safer to open the radial bursa.

If the radial bursa is involved, the site of the greatest tenderness is along the course of the *flexor pollicis longus*, beneath the ulnar border of the short muscles of the thumb.

Treatment of Infections of Radial and Ulnar Bursæ.—If these infections are secondary, as they usually are, to infections of the related tendon sheath, it is easy to pass a probe-pointed, grooved director up the sheath and open the bursa upon this. In infection of the ulnar bursa the incision is made on the ulnar side of the bursa, so that the skin incision, lying, as it does, to the ulnar side of the flexor tendons, does not interfere with the mesenteries and their contained blood vessels which enter on the radial side. Moreover, being on the radial side of the hypothenar area, this incision enters the deepest part of the ulnar bursa. After the pus has been sponged away, pressure over the area of Parona's space, if this space is infected, will make pus appear in the opened sheath. If Parona's space is infected, it is opened and drained. In draining the radial bursa the skin incision should run along the ulnar border of the thenar muscles, and the medial border of these muscles is followed till the swollen sheath of the radial bursa is recognized. It is most important that the position of the branch of the median nerve to the thenar muscles be remembered. To avoid it with safety, do not prolong the incision further proximally than 2·5 centimetres (one inch) distal to the transverse carpal ligament.

Infections of the ulnar bursa secondary to infections of the radial bursa, but not involving the tendon sheath of the little finger, may be opened by an incision in the distal third of the palm, over the usual site of the greatest tenderness in ulnar bursa infections. On careful dissection with the aid of tourniquet the tense bursa will be seen to herniate into the wound and may readily be opened.

The upper ends of both the radial and ulnar bursæ are best drained by the incisions recommended for drainage of Parona's space.

In all tendon sheath and bursal infections care should be taken when opening not to injure the tendon itself; and drainage material should not be placed in the space of the sheath, but should extend only to the sheath.

Pieces of rubber glove are recommended for drainage of these spaces, but even these should not be left in longer than forty-eight hours. Post-operative treatment consists of hot saline baths or voluminous foments. After heat has been discontinued, dress with vaseline gauze, as this can be easily and almost painlessly removed.

Lymphangitis.

The lymphatics of the palmar aspect of the hand and fingers are divided into two sets, the superficial and the deep. The superficial group form a very close and rich meshwork, the efferents of which run to the dorsum of the hand by the shortest possible route; and this is the explanation of the marked dorsal swelling of the hand which usually accompanies palmar infections. One efferent which calls for special mention runs from the palm of the hand through the palmar fascia and over the transverse head of the adductor muscle, so coming to lie on the first dorsal interosseous muscle, and then joins with the dorsal lymphatic collectors. The superficial plexus of the lymphatics lies superficial to the venous plexus and its efferents accompany the superficial veins. The efferents from the innermost two, possibly three, fingers end in the supratrochlear gland. While the most external efferent runs in the deltopectoral space and ends as a rule in the subclavian gland lying on the junction of the cephalic

and axillary veins, sometimes it may be seen to pass over the clavicle and to drain into the supraclavicular glands.

These anatomical facts make lymphangitis of the external portion of the hand a much more dangerous clinical condition than an equally severe infection of the innermost portion of the hand. Some of the external efferents may also run across the anterior surface of the forearm and drain into the supratrochlear gland. The deep lymphatics run along with the brachial artery and its chief branches and so drain the subfascial portions of the hand. At intervals they communicate with the superficial lymphatics, and most of them drain into the humeral group of the axillary glands, a few end in the supratrochlear gland, and others terminate in the paravascular nodes found along the course of the deep vessels. Lymphangitis may be either superficial or deep. It may follow some slight injury, for example, a pin prick or a splinter, or some superficial abrasion, or deep infection of the hand. It is most commonly due to invasion by streptococci, of which the hæmolytic group are the most virulent and pathogenic.

In deep lymphangitis the course naturally follows the pathway of the vessels. Exceptionally abscesses form along the course of these vessels, either in the paravascular nodes or in the "lakes" into which the lymphatics widen at irregular intervals.

Signs and Symptoms.—The site of infection is generally surrounded by an area of redness and swelling which is hot and tender, but the local changes may be so slight as to escape notice were it not for the accompanying pain. Even pain is not an invariable symptom.

If the finger is the site of the primary infection, the whole finger may be stiff and swollen. The dorsum of the hand and forearm show red lines which are the inflamed lymphatic vessels draining the site of the primary infection. The lumen of the lymphatic vessels contains bacteria and fibrous clots, the endothelial lining may be destroyed and the wall infiltrated with cells and, at times, bacteria. The supratrochlear and axillary glands may be swollen and tender. The systemic reaction varies within wide limits with the virulence of the infection and the resistance of the patient. The signs of toxæmia are early evident: high or low temperature, headache, anorexia and prostration. The treatment of lymphangitis is passive; rest and heat are the two most useful aids in stimulating the body's resistance. Heat is best supplied in the form of hot hypertonic saline baths, but if these are not practicable, hot voluminous fomentations accomplish much. Rest in bed is essential. Light diet, plenty of fluids and eliminatives are important subsidiary aids. In the more fulminating cases antistreptococcal serum in thirty to fifty cubic centimetre doses, given subcutaneously, may be of some avail, but generally they are not needed. Passive hyperæmia in very toxic cases appears at times to be beneficial, and one feels that more use should be made of this in the severest types. Abscess formation is the most frequent complication of lymphangitis affecting the hand, and they are found to occur most commonly at the following sites: in the tendon sheath, on the dorsum of the hand, just above the anterior ligament on the volar surface, in the supratrochlear, axillary and supraclavicular glands. These local abscesses may occur in about 10% to 15% of cases (Kanavel).

Kanavel has emphasized the intimate relation existing between the lymphatics of the fingers and the tendon sheaths, and it should be a cardinal rule that in all cases of lymphangitis constant watch be maintained for involvement of the associated tendon sheaths.

Age has an important bearing on the severity of lymphangitis, the prognosis after middle age being much less favourable.

Thrombophlebitis is another serious complication of lymphangitis and frequently leads to septicæmia and death.

In lymphangitis active treatment by incision is nothing short of a calamity, ill-advised incision frequently leading to an overwhelming toxæmia and death. It should be a rule that no incisions be made unless in the presence of localized pus, for example, in tendon sheath infections or abscess of the dorsum of the hand.

Cellulitis.

In the palm of the hand there are three fascial spaces, the hypothenar, thenar and middle palmar spaces. Infections of the hypothenar have already been discussed and are in no way as serious as infection of the latter two sites. In the normal hand these spaces are filled with loose areolar connective tissue, the packing tissues. The thenar space is an L-shaped space lying superficial and distal to the transverse head of the *adductor pollicis*; it extends medially as far as the middle metacarpal bone, and dorsally as far as the volar surface of the metacarpal bone of the index finger. The adductor muscle is covered over by a condensation of the areolar tissue of the space forming a layer of deep fascia. The volar boundary of the space is a dense layer of tissue which fuses with the palmar fascia, and in this tissue run the tendon and lumbrical muscle of the index finger. The radial bursa also lies superficial to the radial side of this space. The radial longitudinal crease of the palm marks the ulnar boundary of the thenar space (see Figure II).

The middle palmar space extends from the middle metacarpal bone to the radial side of the metacarpal bone of the little finger. A layer of deep fascia lying on the volar interosseous muscle forms the dorsal boundary, and on its volar side it is separated by another condensation of areolar connective tissues from the lumbrical muscles, the tendons of the little, ring and middle fingers, and from the ulnar bursa. On its radial side it is limited by a dense fibrous septa, attached to the middle metacarpal bone, which separates it from the thenar space in such a way that neither one communicates with the other (see Figure IV).

FIGURE IV.

The middle palmar space and its lumbrical extensions. The transverse ligament of the carpus has been divided in part to expose the space more clearly. (After Hughes-Jones.)

Both spaces come into relation with the lumbrical muscle and tendon sheath of the middle finger, but Kanavel has shown by means of experimental injections that the tendon sheath of this finger more easily ruptures into the middle palmar space. So the middle palmar space

is in relation with the lumbrical muscles and tendon sheaths of the little, ring and middle fingers, while the thenar space is in relation with the lumbrical muscles and tendon sheath of the index finger. This relationship is indirect, by way of what Kanavel calls the loft, which is a mass of loose connective tissues superficial to the major spaces. A line drawn across the palm joining the radial end of the proximal transverse palmar crease with the ulnar side of the distal transverse palmar crease corresponds to the distal limit of these major fascial spaces, while a line drawn across the palm 2·5 centimetres (one inch) distal to the distal transverse crease of the wrist marks their proximal extent.

Source of Infection of the Fascial Spaces.—The major fascial spaces are most commonly infected by direct spread from the associated tendon sheaths, lumbrical spaces and bursæ, or by direct implantation, or following upon infections of the metacarpal bones, for example, following infected compound fracture of the bones. Infection may also follow a distal palmar hour-glass abscess, spreading up along the lumbrical canals. In delayed radial and ulnar bursal infection the direction of spread is usually to Parona's space, but infrequently the palmar cellular spaces may be involved. Lastly, infection may be carried to these spaces by the lymphatics, and in this regard the deep lymphatic vessel which runs from the palm across the transverse head of the adductor muscle is very important. I have under my care at present a patient who had a superficial palmar abscess which was inadequately opened. When I first saw him there was a draining wound in the palm of the hand, which was greatly ballooned, due to infection of the thenar space. On drainage of the latter the swelling quickly disappeared.

Symptoms and Signs.—The difficulty with infections of the major fascial spaces of the palm of the hand is diagnostic rather than therapeutic. The diagnosis is easier if the radial and ulnar bursæ are not involved. Frequently it is only after making an exploratory incision that one can be sure that the palmar spaces are not infected. If this is done aseptically, fresh instruments being used, no great harm follows. Operating with a tourniquet applied and applying pressure carefully over the palmar spaces after the bursæ have been incised often obviate this necessity.

In these infections constitutional reaction is as a rule severe, while locally there is marked swelling, but the hand is not red, but rather whitish, due to the pressure of the underlying inflammatory reaction causing obliteration of the vessels of the skin.

No localized point of extreme tenderness is present, but rather a diffuse tenderness over the whole area involved. There is marked swelling of the palm, with obliteration of the palmar concavity, which is replaced by a palmar convexity, a ballooning of the palm.

This ballooning is much more marked in the thenar space infections than in infections of the middle palmar space, owing to the fact that over the former the palmar fascia is thin as compared with the thick, strong, central part which overlies the latter. In thenar space infection the thumb is abducted so as to leave the adductor muscle relaxed, thus reducing as far as possible the tension in the space. Movement of the terminal phalanx of the thumb is comparatively painless, which is of great diagnostic importance in differentiating thenar space abscess from infections of the radial bursa. In middle palmar space infections loss of function of the hand is variable, but if the tendon sheaths are not involved, the patient is able and willing to move his fingers and does not feel the excruciating pain on passive extension which, as we have seen, is so characteristic of infections of the palmar bursæ. It should be remembered that in patients who present themselves for treatment several days after the onset of infection, the pain and tenderness in the hand may not be very great, owing to the blood supply to the nerves being obliterated by the pressure of œdema, with consequent loss of their function.

From these palmar space infections the pus frequently spreads along the diverticula accompanying the lumbrical muscles and comes to point on the dorsum of the web. The middle palmar space may be opened by incising the web between the middle and ring fingers or between the ring and little fingers, depending upon the source of infection. The lumbrical space is thus opened. A blunt-nosed artery forceps is then pushed along the lumbrical muscle a thumb's breadth and a half into the middle palmar space, opened, and the pus is evacuated. The incision may be kept open for a few days by a piece of rubber glove inserted into the space. Another method of draining these spaces is to make an incision in the web and expose the lumbrical muscle. The incision is continued into the palm for 2·5 to 3·75 centimetres (one to one and a half inches). This brings the incision between the tendons. An artery forceps is thrust under the group of palmar tendons into the abscess cavity. The thenar space is drained either by an incision on the posterior surface of the web and parallel to its free border or through an incision parallel to the radial border of the metacarpal of the index finger and opposite its middle two-fourths on a level with its volar surface. A blunt-nosed artery forceps is pushed through the incision and is passed across the volar surface of the index metacarpal between the adductor transverse muscle posteriorly and the short muscles of the thumb and flexor tendons anteriorly (see Figure V).

FIGURE V.

From Kanavel's "Infections of the Hand", for comparison with Figures II and IV. Drawing showing the drainage under tendons. AT, *adductor transversus*; LS, lumbrical space; MPS, middle palmar space; TS, thenar space; UB, ulnar bursa.

When opening either fascial space, great care should be exercised not to push the point of the forceps beyond the middle metacarpal bone, for if this is done, the other major fascial space will be entered, with possible extension of the infection to this area.

Infection of the Major Forearm Space, Parona's Space.

Parona's space is the fascial space which lies between the *flexor profundus digitorum* and *flexor pollicis longus* in front, and the *pronator quadratus* and interosseous membrane behind. On either side, on a level with the volar surface of the radius and ulnar, it comes to be very superficial, being separated from the skin only by the deep fascia joining the volar muscles to the bone.

Infection generally occurs as a complication of infection of the ulnar or radial bursa, rarely as a complication of infection of the middle palmar space.

Diagnosis is based on increased swelling of the forearm, induration and tenderness at this site, but as the incisions for drainage of this space also give excellent drainage to the proximal portion of the radial and ulnar bursæ, there should be no hesitation in making them. An incision 6·75 to 7·5 centimetres (two and a half to three inches) long is made on the volar surface of the ulna, about 3·75 centimetres (one and a half inches) above the ulnar styloid process. The deep fascia is divided, an artery forceps is pushed across behind the flexor tendons and, if considered necessary, a second incision on the radial side is made by cutting down on the artery forceps; this method of making the radial incision insures against injury of the radial artery. If the infection is secondary to a bursal infection, the forceps may be plunged into the proximal end of the sheath and opened, or the sheath may be opened by the finger. A rubber glove drain is introduced transversely across the forearm, just in front of the bones and the *pronator quadratus* muscles. It is removed in forty-eight hours.

Summary.

In all infections of the hand an accurate and correct diagnosis is the basis of successful therapy. Incisions should avoid creases and, when possible, digital vessels and nerves. The pus should be examined microscopically and cultured. An exact knowledge of the type of organisms is helpful in treatment and often aids in a more exact prognosis.

The importance of rest cannot be stressed too often or too much. A splint applied to an infected finger gives the patient a feeling of security, protects the part from further injury, but, more important, it prevents movement and so stops the associated milking action in the lymphatic and venous systems.

Therefore the essential rule in the treatment of these infections is rest, and if the patient shows signs of systemic reaction, such as a high temperature, he should be confined to bed, bodily rest being thus insured.

After rest, heat is one of the most useful aids. It gives the patient relief from pain and aids Nature in its fight against the invading organisms. It may be used in the form of hot hypertonic saline baths or large voluminous fomentations frequently changed.

After the temperature has fallen, hot baking in the arthritic baker or medical diathermy is a useful therapeutic agent.

In all infections of the hand one of the surgeon's most important duties, more particularly in severe infections, is to see that the hand is kept in the "position of function", namely, dorsally flexed at the wrist, slight flexion of the fingers, the fingers abducted, and the thumb abducted with its palmar surface facing the palmar surface of the fingers. A lightly padded aluminium splint, a rolled bandage or a plaster cast is a useful aid in maintaining this position.

As in treatment of all severe infections, there is no need to stress here the value of plenty of fluids, given by mouth and bowel preferably, but, if necessary, intravenously. As many of these patients have septicæmia, subcutaneous administration of fluid is not advisable, on account of the risk of lowering resistance at the site of the injection, with resulting pyæmic abscess.

The diet should be light, the bowels should be kept open, the kidneys active. Once the temperature has fallen the tonic effect of sunlight should be remembered and utilized. In very ill patients antistreptococcal serum and Bier's bandage hyperæmia are useful aids, but the greatest reliance must always be placed on rest and heat.

As soon as the temperature has dropped, encourage active movements.

Early movement is the third desideratum in the treatment of these infections.

General anæsthesia, preferably by nitrous oxide and oxygen, is essential. Infections of the hand are not suitable for local anæsthesia and many tragedies have followed its use. In operating on these patients a well applied tourniquet should always be used, and for this a blood pressure armlet is ideal. Incisions should be adequate both as regards length and depth. They should be kept open by rubber drain or vaseline gauze, never by dry gauze, which comes to act only as a plug, nor by new stiff rubber tubes, on account of the risk of their causing sloughing and necrosis. All dressings after operation should be done under the most aseptic conditions. When the drains have been removed, vaseline gauze should be used as a dressing because of the ease with which it can be removed.

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British Medical Association News.

SCIENTIFIC.

A MEETING OF THE NEW SOUTH WALES BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Royal Prince Alfred Hospital, Camperdown, on May 30, 1934. The meeting took the form of a series of demonstrations by members of the honorary staff. Part of this report was published in the issue of August 18, 1934.

Neuro-Surgical Cases.

The following neuro-surgical cases were presented by the Surgical Professorial Unit (PROFESSOR H. R. DEW and DR. R. A. MONEY).

Meningioma from the Transverse Sinus and Tentorium Cerebelli.

The first patient was E.H., a female, aged twenty-four years, who was admitted to hospital on April 10, 1934, with a history of headaches above the eyes, especially the left, for six or seven years. The headaches had been much worse for six months. Occasional attacks of vomiting had occurred with relief of the headaches. About two months previously all the teeth had been extracted under general anæsthesia, shortly after which her eyesight became dim and she noticed numbness of the left side of her face and buzzing in the left ear. She had rapidly become blind in the left eye and on admission to hospital could only just distinguish light in the right eye.

Examination of the cranial nerves revealed bilateral anosmia, advanced bilateral papilloedema, with dilated pupils sluggishly reacting to light, and a marked paresis of both lateral recti muscles. There was hyperæsthesia and paresis of the left side of the face and the left cornea. The patient's amaurosis soon become complete in both eyes. The remainder of her nervous system was intact. X ray films of the skull revealed a large calcareous shadow above the tentorium in the left occipito-temporal area. The pre-operative diagnosis was oligodendroglioma of the occipital lobe, on account of the calcification and her age, although the possibility of a meningioma was also considered.

Operation was performed on April 17, 1934. A parieto-occipital osteoplastic flap was raised on the left side under paraldehyde, local infiltration and intratracheally administered ether anæsthesia. A large meningioma was found growing from the angle of the transverse sinus and the *tentorium cerebelli* and displacing the brain, with much deformity.

About half this tumour was removed by endothermy and traction methods, but much hæmorrhage was encountered from large vessels, and two blood transfusions had to be given. The wound was closed without drainage.

During the next two weeks the patient's condition improved in every way except the amaurosis.

On May 9, 1934, at the second operation, the remainder of the huge tumour was removed by endothermy. The lateral ventricle was opened and the tumour was found to be embedded in the brain almost to the mid-line, as far forward as the *incisura cerebelli*. The site of its origin on the transverse sinus and tentorium was coagulated.

Another blood transfusion was given and the wound was closed. Except for some escape of cerebro-spinal fluid for forty-eight hours, convalescence was uneventful and the wound healed perfectly. All her other symptoms had been relieved, but optic atrophy remained and it was doubtful whether any perception of light would be regained.

The tumour was exhibited, showing the two large main masses and the scallops removed with the electric loop. After dehydration the tumour weighed 140 grammes; it must have been about 200 grammes *in situ*. A microscopical section of the tumour was also exhibited, showing a typical meningeal growth.

X ray films illustrating the calcareous area were also shown.

Hæmangioblastoma of the Frontal Lobe.

The second patient, a male, aged twenty-eight years, had been admitted to hospital in March, 1933, under the care of Dr. Eric Susman.

Early in September, 1932, the patient "fainted" whilst in the street, but was able to walk home afterwards. Thereafter he had several such attacks, during which he lost consciousness for about fifteen minutes; the right side of his face and tongue would twitch and then the right upper and lower extremities. After each attack he would feel dazed for several hours.

In February, 1933, he developed severe generalized headache and vomited frequently. In March, 1933, he noticed difficulty in speaking and hesitation in finding the right word to use. His memory became very impaired; at times he would become very quiet and morose, and at other times very irritable. His eyesight was failing rapidly.

On examination he had rather a dull, vacant expression; he was partially aphasic and would hesitate a long time before speaking, and then was often unable to find the right word.

The right side of his face and tongue showed definite paresis and his power in the right hand was impaired. The fundi showed papillœdema, more marked on the left side. There was a generalized diminution in his fields of vision. All other investigations revealed no abnormality. Operation was performed on March 29, 1933, when a large fronto-parietal bone flap was turned down under local infiltration anæsthesia after bromide premedication. The precentral gyrus and the posterior ends of the inferior and middle frontal gyri were very broad and bulged considerably. An incision was made into the most prominent part with an endothermy knife, and a tumour was encountered at a depth of about one centimetre. The presenting solid portion of this tumour was excised and a large cyst opened into, which extended into the frontal lobe and towards the insula. Its contents were evacuated and as much as possible of the tumour was removed. Hæmostasis was secured and the cavity was filled with saline solution. After the operation the patient was completely aphasic for one week, but thereafter convalescence was uneventful, except for a mild infection of the wound behind the ear. Biopsy showed that the tumour was probably a hæmangioblastoma or an angioblastic meningioma, a slide of which was exhibited. In April and May, 1933, he was given a full course of deep X ray therapy to the left fronto-parietal region.

After the operation his aphasia and hemiparesis improved, his headache disappeared and his eyesight and fundi gradually returned to normal.

He had one definite "fit" in December, 1933, and occasionally he has experienced "funny" feelings and paræsthesia in his tongue, mouth and right side of the face, also the right forearm and hand. The infection in the lower posterior corner of the wound resulted in a mild degree of osteomyelitis and a discharging sinus, and on two occasions small areas of bone had to be removed; otherwise he felt well and was anxious to return to work.

Bone Graft to the Skull.

In August, 1932, R.G., a male, aged fourteen years, was kicked by a horse and sustained a compound, comminuted, depressed fracture of the left frontal bone. He was treated by Dr. W. Cletus Smith, of Coraki, who removed the loose and depressed fragments of bone from the brain, together with lacerated *dura mater* and cerebral tissue. The remainder of the depressed area was elevated; the scalp wound was closed by an immediate suture and healed by primary intention. The patient made an uneventful recovery. No symptoms of intracranial mischief followed, but he developed a mild degree of *hernia cerebri* and was given a metal plate to wear inside his cap. This made him a source of unusual interest to his family and playmates and produced rather a bad psychological influence on the boy, who could not play games *et cetera* like his fellows for fear of injuring his brain. On examination in March, 1934, the scar of his injury was visible in the left frontal region and there was an oval gap in the cranium, about six by four centimetres in size, in which the cerebrum was herniating somewhat and pulsating. No evidence of involvement of his central nervous system was detected.

On April 16, 1934, the scalp was turned down from over the defect in the left frontal bone, the newly formed *dura mater* was carefully dissected off it and the edges of the bone were freshened. A piece of bone, cut to the correct size, was removed from the inner table of the right iliac bone and grafted into the gap and held in place by four sutures of chromicized gut. Both wounds were closed without drainage and, except for a small collection of blood and serum which required to be evacuated, healed by primary union. The new graft appeared to have united well already and no pulsation could be detected in his left frontal region. An X ray film showing the defect in the frontal area was exhibited.

B.M., aged forty-one years, on whom a similar operation had been performed two and a half years ago, as reported in THE MEDICAL JOURNAL OF AUSTRALIA of August 27, 1932, was also presented to show that the graft had not disappeared in that time.

Facio-Hypoglossal Nerve Anastomosis.

O.D., a male, aged forty years, in March, 1933, sustained a fracture of the base of the skull when a heavy pole fell on his head. He was unconscious for one week. On recovering, he was found to have complete paralysis of the left side of the face and to be deaf in the left ear. These conditions had both persisted and, in addition, the muscles of the left side of his face had wasted and his eye watered a lot. He suffered from headaches at times, with persistent tinnitus and some weakness of the legs. When examined in May, 1933, he was found to have a complete lower neurone lesion of his left seventh and eighth cranial nerves with atrophy of all the muscles supplied by the seventh nerve. No galvanic responses were obtained. X ray examination of his skull, with special reference to petrous bone, showed no evidence of fracture.

On July 20, 1933, an anastomosis was performed between the twelfth nerve proximally and the seventh nerve distally in the neck, on the left side. Convalescence was uneventful. He was given a splint to support the angle of his mouth. In January, 1934, his face appeared to be more symmetrical in repose; the left side of his tongue had atrophied. In March, 1934, very slight movements were noticed in the muscles of the left side of the face in swallowing, and the corner of the mouth was definitely higher.

In May, 1934, the patient could voluntarily produce some movements at the angle of his mouth and chin, and some "flickering" was noticed in the lower eyelid.

In view of the long period which had elapsed between the accident and the time of anastomosis, the fibres of the facial nerve and the muscles had completely degenerated and the result was accordingly regarded as promising. Further improvement should occur.

Foreign Body (Bullet) in Spine with Partial Division of the Spinal Column and Nerve Roots.

N.McD., a male, aged twenty-seven years, was shot under the left arm about June, 1933, the bullet entering near the anterior axillary fold and reaching the vertebral column in the neck. On admission to another hospital he was found to have tetraplegia, with incontinence of urine and fæces. This condition persisted for several weeks.

Laminectomy was performed on two occasions, but the bullet could not be removed. Gradually, however, power

returned in his right upper extremity and in both lower extremities, more slowly and less completely in his left upper extremity. After four months he could walk, but in May, 1934, his gait was not steady, and his left arm, forearm and hand were weak and wasted. He had also noticed a numbness of the right side of his body, including the upper and lower extremities.

On examination the scars of the wound of entry could be seen in the left axilla, also of the previous laminectomies in the lower cervical and upper dorsal regions. The left upper extremity showed marked wasting and paresis of the triceps, flexors of the wrist and fingers, and all the small muscles of the hand, and there was loss of both protopathic and epicritic sensibility along its medial half in the skin areas supplied by the nerve roots of the eighth cervical and first and second thoracic cord segments, which also innervate the above muscles. Sensation to deep pressure was only slightly affected. Trophic changes were noticeable. The lower left extremity showed some wasting of the quadriceps and calf muscles. His gait was unsteady and the left leg appeared to be thrown forward at the knee, due to some foot-drop. There were no sensory changes, but there was an extensor plantar response on the left side and the knee jerks were increased.

There was definite analgesia and loss of protopathic sensation along the medial half of the right upper extremity, over the right side of the thorax, below the second thoracic segment and trunk, including the right testicle, and over the whole of the right lower extremity. Epicritic sensibility was only slightly impaired in these areas. The left upper limb felt colder than the right, and there was a complete Horner's syndrome seen in the left eye.

Diagrams showing the distribution of the sensory changes and X ray films showing the bullet embedded in the pedicle of the sixth cervical vertebra were shown. Apparently the bullet had damaged the spinal nerves, eighth cervical and first and second thoracic, on the left side, together with their white rami, to the inferior cervical ganglion of the sympathetic, and then the left lateral fasciculus of the spinal cord containing the spino-thalamic, spino-cerebellar and lateral cortico-spinal tracts.

While the patient was in hospital a Queckenstedt test had been carried out and lipiodol injected by a *cisterna magna* puncture, both of which procedures failed to reveal any evidence of a block or pressure caused by the bullet. In view of these facts and the gradual improvement of the patient, it had been decided not to operate again at present, but to persist further with physiotherapy for the paralysed muscles. It was not considered likely that any change would take place in the crossed anæsthesia.

Cervical Rib and Neurogenic Sarcoma of the Ulnar Nerve and Eighth Cervical Spinal Nerve.

About two years ago, A.B., a male, aged eighteen years, first noticed pain and tiredness in the right upper extremity after carrying weights or wearing a heavy coat. Then tingling appeared along the inner border of the hand and forearm and the grip of the right hand became weak. The pains had become much worse and more constant lately, especially at night, and the hand was practically useless.

When admitted to hospital in March, 1934, he had an advanced paresis of the small muscles of his right hand and sensory loss in the distribution of the ulnar nerve. An X ray film showed a small cervical rib on the right side, which, with its fibrous band, was regarded as causing a pressure neuritis. There was an area of tenderness just below the clavicle median to the coracoid process, regarded as a local interstitial neuritis of the medial cord of the brachial plexus, from the lesion above. The axilla was clear.

At operation on March 8, 1934, three centimetres of the *scalenus anterior* muscle were resected, allowing the subclavian artery and lower trunk to fall forward and relieve the pressure. The cervical rib and fibrous band were felt but left *in situ*. No relief was obtained from this procedure, and his symptoms progressed until there was evidence of median nerve involvement too. A painful swelling could then be felt below the clavicle and a tender, mobile tumour appeared in the axilla, continuous with the swelling near the coracoid process. The arm became congested when hanging down.

A second operation was performed on May 17, 1934, when a large tumour was found growing in the ulnar nerve and involving the median nerve too. The axillary vein was almost completely compressed. Complete excision was not possible and a small portion was removed for biopsy, which revealed a spindle-celled fibro-sarcoma of the ulnar nerve. In view of the malignancy of this tumour and its extent and the destruction of several nerves of the brachial plexus, and the advent of a useless, painful arm, an interscapulo-thoracic amputation was undertaken on May 24, 1934. The tumour was found to continue up as far as the intervertebral foramen for the eighth cervical nerve and had to be cut across there.

It was stated that a course of deep X ray therapy had been started to prevent recurrence, but the prognosis was not good and the growth would probably extend inwards to cause a compression myelitis. The dissected specimen of the tumour and a microscopic slide of its structure were exhibited.

Wound of Radial Nerve and Biceps Brachii Muscles.

On March, 30, 1934, A.J., a male, aged nineteen years, injured his left arm and forearm when he put his left upper extremity through a window. When examined, he had a deep incised wound, about seven centimetres long, across the antero-lateral aspect of his left arm, about four centimetres proximal to the elbow joint, and several smaller wounds of the forearm and hand. He was too much under the influence of alcohol to allow a test of his voluntary movements and sensations to be made.

Under ether anæsthesia the wound in his arm was explored, and it was found that the *biceps brachii* muscle had been cut almost completely across, the *brachio-radialis* muscle had been severed at its origin, and also some fibres of the *extensor carpi radialis longus*. The radial nerve had been partially cut through, about three-fifths of its fibres remaining intact. The nerve was repaired with a fine catgut suture; the wound was cleaned out and the edges were excised; it was then closed with interrupted sutures of horsehair, without drainage. The muscles were not sutured. The other wounds were similarly dealt with and an unpadded plaster cast was applied to the arm, forearm, hand and fingers, to keep the elbow flexed at 90° and the wrist and fingers extended fully. Three days later it was noticed that the patient could extend his fingers actively and sensations were intact, except in the little finger, which was practically anæsthetic, due to a wound on the dorsum of the hand. The patient was discharged from hospital. The plaster cast was removed four weeks later. All the wounds had healed perfectly and all movements were present. His power had fully recovered. He had had no massage or electrical treatment. The patient was presented to show the value in cases similar to this of the use of the unpadded plaster cast in saving the expense of hospitalization, dressings and physiotherapy.

CHAMPIONSHIP GOLF MEETING.

THE first annual championship golf meeting of the Victorian Branch will be held on Thursday afternoon, September 27, on the Royal Melbourne Golf Links, Cheltenham.

A perpetual challenge cup has been offered by the President of the Branch, Dr. Gerald Weigall, which will be held by the winner for one year, and a miniature cup, suitably engraved, will also be presented to the winner.

Concurrently with this event will be a handicap stroke event for a perpetual trophy and a miniature cup. This cup has been offered (conditionally upon no other being forthcoming) by a non-player. The Committee is grateful for this happy gesture and is ready to accept it, but it is the donor's desire that such a presentation should first be open to a golf player.

The trophies will be awarded at the annual meeting on December 5. Entrances close on September 20, 1934.

NOMINATIONS AND ELECTIONS.

THE undermentioned has been elected a member of the New South Wales Branch of the British Medical Association:

Sheehan, William Beverley James, M.B., B.S., 1927 (Univ. Sydney), 273, Cleveland Street, Redfern.

Correspondence.

REDUCED TELEPHONE CHARGES TO DOCTORS.

SIR: In the issue of August 11 Dr. Stevenson pleads for a reduction in telephone charges for doctors.

Might I mention that I have recently discovered that doctors are charged a pound yearly more than the ordinary individual if they wish their name to appear in the book as "Dr."?

I can have "Mrs." (a letter more) without this extra charge.

I have a professional, a private house, and a seaside house telephone, and because I have "Dr." in the book I pay three pounds a year extra rental.

Perhaps the Department might be induced to treat us as ordinary mortals.

Yours, etc.,

J. G. EDWARDS.

185, Macquarie Street,
Sydney,
August 10, 1934.

Post-Graduate Work.

ANNUAL COURSE IN OBSTETRICS IN MELBOURNE.

THE Melbourne Permanent Post-Graduate Committee announces that the annual course in obstetrics will be held this year at the Women's Hospital, Melbourne, during the fortnight Monday, September 24, to Saturday, October 6, inclusive. The course comprises attendance at the routine work of all departments of the hospital, as well as special demonstrations and lectures by members of the honorary staff. Arrangements have been made whereby a limited number of graduates can reside at the hospital during the course. The fee for the course is three guineas. An additional charge of three guineas per week is made for board at the hospital. It is requested that those proposing to attend this course and those wishing to take advantage of hospital accommodation should send their names and fee as soon as possible to the Honorary Secretary, 61, Collins Street, Melbourne, C.1.

INTERNATIONAL MEDICAL POST-GRADUATE COURSES IN BERLIN.

THE following international medical post-graduate courses have been arranged for October, 1934, by the Berlin Academy for Medical Post-Graduate Training, under the auspices of the Lord Mayor of the City of Berlin and in succession to the Society of Lecturers for Medical Post-Graduate Training in Berlin:

1. Internal medicine, with special regard to tuberculosis, from October 1 to 13. Fee: 60 RM.
2. Course in tuberculosis, in the tuberculosis hospital of the City of Berlin, from October 15 to 20. Fee: 50 RM.
3. Obstetric-gynæcological post-graduate training week from October 15 to 20. Fee: 50 RM.
4. Post-graduate course in diseases of the ear, nose and throat, from October 1 to 13. Fee: 120 RM.
5. Post-graduate course in pædiatrics, from October 22 to 27. Fee: 50 RM.
6. Surgical intrathoracic diseases, with special regard to pulmonary tuberculosis, from October 29 to November 2. Fee: 80 RM.
7. Individual courses in all branches of medicine, with bedside and laboratory practice, are held every month. The fee is 50 to 80 RM for eight lessons of two hours each. In these courses special attention is paid to practical work; theory plays a minor part.

Programmes and further particulars obtainable from the Berlin Academy for Medical Post-Graduate Training, Berlin NW 7, Robert Koch-Platz 7 (Kaiserin Friedrich-Haus).

Foreign students attending the courses obtain a reduction of 60% in fares from the German Railways.

Analytical Department.

"EXORCIN."

"EXORCIN" solution is a solution of 1 in 1,000 hexyl resorcinol. It is claimed to be a powerful non-poisonous tissue disinfectant adjusted to a low surface tension of 37 dynes per centimetre. The action of hexyl resorcinol is well known.

A sample of "Exorcin" was bought in the open market and submitted to our analyst. He reports that the liquid in full strength kills typhoid bacilli in fifteen seconds. The carbolic acid coefficient is $^{15}/_{100}$ = 0·15, that is, a dilution of "Exorcin" of 1 in 15 of water is equivalent from the bactericidal point of view to a 1 in 100 dilution of carbolic acid.

"Exorcin" may be recommended as a reliable antiseptic.

Naval, Military and Air Force.

DINNER IN HONOUR OF MAJOR-GENERAL G. W. BARBER, C.B., C.M.G., D.S.O.

A DINNER was given by the Australian Army Medical Corps Officers' Mess to Major-General G. W. Barber on Saturday, August 4, 1934, at the Naval and Military Club, Melbourne, to bid him farewell on the occasion of his retirement from the position of Director-General of Medical Services, Commonwealth Military Forces.

His successor, Colonel Rupert M. Downes, was in the chair, and there was a large gathering of Army Medical Corps officers, senior and junior, including representatives from other States. Major-General J. H. Bruche, Chief of the General Staff, was present as a guest, and there were other representatives from the combatant and medical services of the Air Force and Navy.

The toast of the health of General Barber was proposed by Colonel Robert Fowler and supported by General Bruche.

The toast of the health of General Barber's successor, Colonel Rupert Downes, was proposed by Major-General R. H. Fetherston and supported by Colonel Victor Hurley.

Honours.

HIS MAJESTY THE KING OF ITALY has been pleased to confer on Dr. G. B. Battaglia, of Brisbane, the honour of knighthood in the Distinguished Order of the Crown of Italy. We tender Dr. Battaglia the congratulations of the members of the medical profession in Australia.

Obituary.

THOMAS EDWIN LLEWELLYN LAMBERT.

We regret to announce the death of Dr. Thomas Edwin Llewellyn Lambert, which occurred on August 9, 1934, at Malvern, Victoria.

EDWARD AUGUSTUS LERMITTE.

We regret to announce the death of Dr. Edward Augustus Lermitte, which occurred on August 15, 1934, at Cleveland, Queensland.

WILLIAM ROBERT TOMLINSON.

We regret to announce the death of Dr. William Robert Tomlinson, which occurred on August 18, 1934, at Orange, New South Wales.

Books Received.

SURGICAL ANATOMY AND PHYSIOLOGY, by N. C. Lake, M.D., M.S., D.Sc., F.R.C.S., and C. J. Marshall, M.D., M.S., F.R.C.S.; 1934. London: H. K. Lewis. Demy 8vo., pp. 898, with 238 illustrations. Price: 30s. net.

Diary for the Month.

AUG. 28.—New South Wales Branch, B.M.A.: Medical Politics Committee.
AUG. 30.—South Australian Branch, B.M.A.: Branch.
AUG. 30.—New South Wales Branch, B.M.A.: Branch.
SEPT. 3.—New South Wales Branch, B.M.A.: Organization and Science Committee.
SEPT. 4.—Tasmanian Branch, B.M.A.: Council.
SEPT. 5.—Western Australian Branch, B.M.A.: Council.
SEPT. 5.—Victorian Branch, B.M.A.: Branch.
SEPT. 6.—South Australian Branch, B.M.A.: Council.
SEPT. 7.—Queensland Branch, B.M.A.: Branch.
SEPT. 11.—Tasmanian Branch, B.M.A.: Branch.
SEPT. 11.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
SEPT. 14.—Queensland Branch, B.M.A.: Council.
SEPT. 18.—Tasmanian Branch, B.M.A.: Council.
SEPT. 18.—New South Wales Branch, B.M.A.: Ethics Committee.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," pages xvi, xvii and xix.

ADELAIDE HOSPITAL, ADELAIDE, SOUTH AUSTRALIA: Medical Registrar.
BRITISH MEDICAL ASSOCIATION, VICTORIAN BRANCH: Medical Secretary.
CHILDREN'S HOSPITAL (INCORPORATED), PERTH, WESTERN AUSTRALIA: Junior Resident Medical Officers.
LAUNCESTON PUBLIC HOSPITAL, LAUNCESTON, TASMANIA: Resident Medical Officers.
MATER MISERICORDIÆ CHILDREN'S HOSPITAL, BRISBANE, QUEENSLAND: Resident Medical Officer.
PERTH HOSPITAL, PERTH, WESTERN AUSTRALIA: Resident Medical Officers.
PRINCE HENRY HOSPITAL, SYDNEY, NEW SOUTH WALES: Junior Resident Medical Officer.
ST. GEORGE DISTRICT HOSPITAL, KOGARAH, NEW SOUTH WALES: Medical Superintendent.
THE BRISBANE AND SOUTH COAST HOSPITALS BOARD, QUEENSLAND: Honorary Clinical Assistant.
THE BROKEN HILL AND DISTRICT HOSPITAL, BROKEN HILL, New South Wales: Resident Medical Officers.
THE WOMEN'S HOSPITAL, CROWN STREET, SYDNEY, NEW SOUTH WALES: Junior Resident Medical Officer.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane Associated Friendly Societies' Medical Institute. Chillagoe Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL are advised, in their own interests, to submit a copy of their agreement to the Council before signing. Lower Burdekin District Hospital, Ayr.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	Combined Friendly Societies, Clarendon and Kangarilla districts. Officer of Health, District Council of Elliston. All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 205, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Editorial Notices.

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